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ENVIRONMENTAL ASSESSMENT BOARD

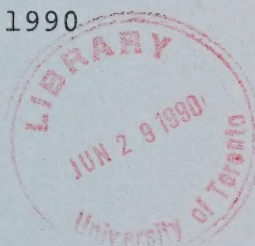
VOLUME: 214

DATE: Wednesday, June 13, 1990

BEFORE:

A. KOVEN, Chairman

E. MARTEL, Member



FOR HEARING UPDATES CALL (TOLL-FREE): 1-800-387-8810

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ASSOCIATES
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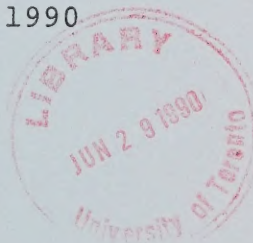
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HEARING ON THE PROPOSAL BY THE MINISTRY OF NATURAL
RESOURCES FOR A CLASS ENVIRONMENTAL ASSESSMENT FOR
TIMBER MANAGEMENT ON CROWN LANDS IN ONTARIO

IN THE MATTER of the Environmental
Assessment Act, R.S.O. 1980, c.140;

- and -

IN THE MATTER of the Class Environmental
Assessment for Timber Management on Crown
Lands in Ontario;

- and -

IN THE MATTER OF a Notice by the
Honourable Jim Bradley, Minister of the
Environment, requiring the Environmental
Assessment Board to hold a hearing with
respect to a Class Environmental
Assessment (No. NR-AA-30) of an
undertaking by the Ministry of Natural
Resources for the activity of timber
management on Crown Lands in Ontario.

Hearing held at the offices of the Ontario
Highway Transport Commission, Britannica
Building, 151 Bloor Street West, 10th Floor,
Toronto, Ontario, on Wednesday, June
13th, 1990, commencing at 8:30 a.m.

VOLUME 214

BEFORE:

MRS. ANNE KOVEN
MR. ELIE MARTEL

Chairman
Member



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I N D E X o f P R O C E E D I N G S

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<u>JOSEPH V. RODRICKS,</u> <u>NANCY J RACHMAN, Sworn</u>	38477
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<u>No.</u>	<u>Description</u>	<u>Page No.</u>
1239	Witness statement of Panel 9B.	38475
1240	Hard copy of overheads to be referred to by Dr. Rachman.	38479
1241	Hard copy of overheads to be referred to by Dr. Rodricks.	38479
1242	Document entitled Notice: Status of Consideration for a Special Review.	38531
1243	Document entitled EPA Updates List of Classified Carcinogenic Pesticides.	38539
1244	Document entitled Mortality Study of Canadian Male Farm Operators: Non-Hodgkin's Lymphoma Mortality and Agricultural Practices in Saskatchewan.	38542
1245	Document entitled The Weight of the Evidence of the Human Carcinogenicity of 2,4-D.	38568
1246	Document entitled Guidelines for Carcinogen Risk Assessment, dated September 24, 1986 published by the U.S. EPA.	38603
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(Cont'd)

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1248	Editorial entitled Herbicides and Non-Hodgkin's Lymphoma, New Evidence From a study of Saskatchewan Farmers by Aaron Blair.	38703

1 ---Upon commencing at 8:30 a.m.

2 MADAM CHAIR: Good morning.

3 MR. CASSIDY: Good morning.

4 MADAM CHAIR: Please be seated.

5 Good morning, Mr. Cassidy.

6 MR. CASSIDY: Good morning. I am not
7 Eleanor Cronk but I am prepared to proceed with the
8 next panel, Panel 9B.

9 Madam Chair, the witnesses are here and I
10 propose to commence by filing a copy of Panel 9B as the
11 next exhibit. Perhaps you could tell me which number
12 that would be, Madam Chair.

13 MADAM CHAIR: Exhibit 1239.

14 MR. CASSIDY: Thank you, Madam Chair.

15 ---EXHIBIT NO. 1239: Witness Statement of Panel 9B.

16 MR. CASSIDY: Madam Chair, the witnesses
17 are present before you. They are Dr. Joe Rodricks and
18 Dr. Nancy Rachman from Environ Corporation. I will ask
19 shortly Dr. Rodricks to outline and give some
20 background to Environ Corporation, but I first wish to
21 advise the Board the reason for these witnesses
22 appearing before you and specifically those reasons are
23 outlined in paragraph 3 of the Executive Summary of
24 Exhibit 1239, the witness statement, which can be found
25 on (ii).

1 Environ Corporation was requested to
2 evaluate and review the relevant evidence before this
3 Board and to advise the Industry and subsequently this
4 Board regarding issues relating to (a) the process by
5 which the environmental protection agency in the United
6 States authorizes pesticides use in forestry as it
7 concerns issues which have arisen in the course of
8 evidence before this Board; (b) the evaluation of
9 public health risks from the use in forestry of
10 pesticides including sources of toxicity and data and
11 risk assessment, and the evaluation of existing
12 scientific evidence concerning the possible toxicity of
13 the pesticides used in forestry and, in particular, Dr.
14 Rodricks will be referring to two major exhibits before
15 this Board dealing with that matter.

16 That is all I propose to say by way of
17 introduction, Madam Chair. I now propose to qualify
18 Dr. Rachman and Dr. Rodricks as expert witnesses and I
19 would ask that Dr. Rachman be qualified as an expert in
20 the registration of pesticides in the United States and
21 Dr. Rodricks be qualified as an expert witness in
22 toxicology, specializing in human health risk
23 assessment principles for chemicals.

24 Both of the witnesses have asked to be
25 affirmed before you this morning.

1 JOSEPH V. RODRICKS,
2 NANCY J. RACHMAN, Affirmed

3 MR. CASSIDY: As I indicated, Madam
4 Chair, I would like to ask Dr. Rodricks to provide a
5 brief description of Environ corporation. For your
6 benefit and the benefit of the parties, a complete
7 description of Environ Corporation can be found at
8 Appendix B of Exhibit 1239. The curriculum vitae for
9 both Dr. Rodricks and Dr. Rachman can be found as well
10 at Appendix B.

11 The description of Environ Corporation
12 commences at page 28 of Appendix B of Exhibit 1239.

13 DIRECT EXAMINATION BY MR. CASSIDY:

14 Q. I would ask, Dr. Rodricks, if you
15 could give a brief overview of that description for the
16 benefit of the Board.

17 DR. RODRICKS: A. Yes, thank you.
18 Environ Corporation is a technical consulting firm with
19 headquarters in Arlington, Virginia. We have offices
20 at three other locations in the United States as well.
21 We are just eight years in this business. We were
22 formed in 1982 to provide risk assessment services to
23 government, to industry.

24 We gathered experts in all of the
25 disciplines related to risk assessment. My background

1 is in health sciences and I direct the health sciences
2 operation of the company. We are mostly toxicologists,
3 epidemiologists, scientists from the public health
4 professions, about half of us, the 40 or so health
5 scientists in the company are at the doctoral level or
6 well beyond that.

7 We also have an even larger staff of
8 environmental scientists and environmental engineers,
9 people who specialize in ground water, in air
10 modelling, physical scientists and engineers, who deal
11 with issues of human exposure to chemicals from many
12 different sources.

13 That staff puts together risk assessments
14 and we have worked on almost every imaginable issue
15 concerning chemicals in the environment or in the work
16 place, occupational exposures as well. We have served
17 also quite a wide variety of clients. We have worked
18 for government in the U.S., for the environmental
19 protection agency on occasion or the Occupational
20 Safety and health Administration. We have also
21 performed consulting of this type for a wide variety of
22 industrial firms, both in the United States and abroad.

23 I think that's a capsule of some of our
24 work.

25 Q. Thank you

1 MR. CASSIDY: Now, Madam Chair, Mr.
2 Martel, these witnessess will be referring to a number
3 of overheads in the course of explaining their evidence
4 this morning and I have prepared hard copies of these
5 overheads which I now provide to the parties and file
6 as the next exhibits.

7 There are two sets of overheads; one set
8 prepared for Dr. Rachman and another set prepared for
9 Dr. Rodricks, and I would ask that they be filed as the
10 next exhibits. I have collected the overheads in a
11 stapled form and numbered -- each overhead numbered and
12 in the course of their evidence, the witnesses will
13 guide you through these overheads by referring to the
14 page numbers on these exhibits.

15 If we could then ask that the set of
16 overheads prepared by Dr. Rachman be the next exhibit,
17 which I believe would be exhibit 1240, and the
18 overheads prepared by Dr. Rodricks be filed as Exhibit
19 1241.

20 I will now provide hard copies of those
21 to the parties and the Board. (handed)

22 MADAM CHAIR: Thank you.

23 ---EXHIBIT NO. 1240: Hard copy of overheads to be
24 referred to by Dr. Rachman

25 ---EXHIBIT NO. 1241: Hard copy of overheads to be
referred to by Dr. Rodrick's

1 MR. CASSIDY: I would like to commence
2 this morning's evidence, Madam Chair, with Dr. Rachman
3 who will be testifying, as is indicated in the
4 Executive Summary, to Section 1 of the witness
5 statement, Exhibit 1239. For the Board's benefit, for
6 your notes, that section commences at page 4 of Exhibit
7 1239 and concludes as at page 23 of the witness
8 statement.

9 As Dr. Rachman will be referring to a
10 number of overheads, I will be going over periodically
11 to turn them on.

12 The first overhead, which could be found
13 at page 1 of Exhibit 1240, will be where we will
14 commence with Dr. Rachman.

15 Q. I would ask you, Dr. Rachman, to
16 please describe for the Board the legal criterion that
17 is used for registration of pesticides in the United
18 States.

19 DR. RACHMAN: A. I'll be happy to do
20 that. Would it be awkward for the Board if I stand?

21 MADAM CHAIR: No.

22 DR. RACHMAN: The registration of
23 pesticides in the United States in under the authority
24 of the Federal Insecticide, Fungicide and Rodenticide
25 Act which is known as FIFRA. Section 3(c)(5) of FIFRA

1 provides the criteria which must be satisfied in order
2 to register a pesticide.

3 What I will do now is to paraphrase for
4 you from the law, rather than quote. In order to
5 register a pesticide, the administrator of the EPA must
6 make a finding that the product's composition warrants
7 the claims that are made for it; that the labeling
8 that's submitted to support the registration, as well
9 as other materials that are required by this section of
10 the law are in compliance with the requirements of the
11 law; that the product will perform its intended
12 function without unreasonable adverse effects on the
13 environment; and that when used according to widespread
14 practice it will not generally cause unreasonable
15 adverse effects on the environment.

16 A couple of definitions here for your
17 information. The term unreasonable adverse effects in
18 the environment is defined to mean an unreasonable
19 risk, taking into account not only risk information but
20 also the economic, social and environmental costs or
21 benefits of the pesticides use. So you will note that
22 this is not a no-risk statute, that there is a risk
23 benefit comparison that is specified by the law.

24 The term environment in FIFRA includes
25 air, water, land and all plants and animals living

1 therein, and also the inter-relationships between all
2 of these living things. So that's a very broad
3 definition of environment.

4 MR. CASSIDY: Q. And those definitions
5 are both contained on page 1 of Exhibit 1240, the
6 overhead?

7 DR. RACHMAN: A. Yes, that's correct.

8 Now, in order to determine whether or not
9 the products for registration meets these criteria, the
10 EPA has the authority require data to be submitted.

11 Q. And that is the subject of the next
12 overhead?

13 A. That's correct.

14 MR. CASSIDY: Which is page 2 of Exhibit
15 1240, Madam Chair.

16 DR. RACHMAN: This overhead summarizes
17 for you the general subject areas. Not the specific
18 tests that are required, but the general subject areas
19 in which data must be supplied and I will tell you in a
20 general way what the EPA is looking for each in each
21 one of these areas.

22 Product chemistry. The registrant must
23 determine the basic physical and chemical
24 characteristics of the active ingredient and a
25 formulated product and must determine what impurities

1 are present. Impurities that are present to a level of
2 a tenth of a per cent or greater must be characterized.
3 If the EPA decides that those impurities are of
4 toxicological significance it has the authority to
5 request the registrant to submit data on those
6 impurities. The registrant must certify the upper and
7 lower limits of active ingredient and impurities in the
8 product as it will be sold.

9 In the area of environmental fate, the
10 idea is to establish the identity of degradates that
11 form when the product is used, and also to characterize
12 in a quantitative fashion the time course of the
13 dissipation of the product in the environment.

14 The toxicology area is the one that
15 pertains to potential human health effects. These are
16 tests of the inherent toxicity of the active ingredient
17 or formulation and there are four general areas
18 included. The short-term testing includes acute
19 toxicity tests and tests of irritation and
20 sensitization. Long-term testing, that category
21 includes chronic feeding, oncogenicity, reproduction.
22 Metabolism is generally a study done in a rat to
23 determine how the pesticide is metabolized in a
24 mammalian system and the identity and quantification of
25 the metabolic products that are formed, and

1 genotoxicity is another word for mutagenicity studies.

2 In the area of wildlife and aquatic
3 organisms, there the intent is to characterize the
4 inherent toxicity to wildlife, non-target organisms,
5 and some of these tests are also done on the formulated
6 product.

7 The residue area involves determining the
8 nature and the magnitude of the residues that are
9 formed in the environment. This is particularly
10 important for pesticides that are applied to food crops
11 or applied to water.

12 Now, there is a broad category called
13 special studies and in here I have lumped several other
14 requirements that may not be imposed on a routine
15 basis, but are required for certain products and
16 certain use pattern. Drift, for example, for certain
17 aerially applied products, specific measurements,
18 actual measurements of exposure under conditions of
19 product use may be required, depending on the
20 additional information that may come to the agency's
21 attention and I will talk a little bit more about that
22 in a minute.

23 The specific tests that are required in
24 each one of these areas are determined in general by
25 the proposed use pattern for the product and

1 specifically the nature of the anticipated exposure
2 from that proposed use pattern. Now, the specific
3 tests are listed -- laid out in the U.S. Code of
4 Federal Regulations, Volume 40. This is referred to as
5 40 CFR.

6 MR. CASSIDY: Which is just at the bottom
7 of that overhead.

8 DR. RACHMAN: Yes, I am sorry, you can't
9 see it, but you will see it on the hand-out.

10 40 CFR, part 158. That part of the U.S.
11 Code of Regulations contains charts and in each one of
12 these subject areas it explains which tests are
13 required depending upon what the proposed use pattern
14 of the pesticide at issue will be.

15 Now, as I've said, the determination of
16 what tests are required is based on the anticipated
17 degree of exposure of the proposed use. In the United
18 States, the food crop use is considered to have the
19 widest potential exposure, the largest number of people
20 are potentially exposed to residues of the pesticides
21 if they are applied to growing food crops and,
22 therefore, in that category, food use is subject to the
23 most extensive data requirements.

24 I call that to your attention because of
25 the chemical pesticides at issue here in this

1 proceeding, six of them are registered for food use in
2 the United States and those are 2,4-D, glyphosate,
3 hexazinone, picloram, simazine and carbaryl. I will
4 return several times to that point and why that is of
5 importance.

6 Now, some requirements are conditional.
7 If you were to go to 40 CFR, part 158 and take a look
8 at those charts that I mentioned, some of the
9 requirements are indicated as conditional. That means
10 that a decision is made by EPA as to whether that
11 particular test is required on a case-by-case basis and
12 those charts also lay out in a series of extensive
13 footnotes the conditions under which those data may be
14 required.

15 I might give you some examples for
16 forestry use pesticides. Worker exposure requirements.
17 The requirement for a specific test of worker exposure
18 to a specific pesticide intended for forestry use will
19 depend on EPA's judgment of the toxicity of the
20 pesticide. They will look at the initial toxicity
21 tests that are required and make the determination as
22 to whether a test of that particular chemical under
23 these conditions is needed.

24 The same thing is true of drift
25 potential. If the pesticide is toxic or if for some

1 other reason the agency thinks that there may be some
2 other potential to drift, maybe because of the physical
3 chemical characteristics of the chemical or the
4 formulation, EPA can require the registrant to do a
5 drift study under actual use conditions.

6 That authority is fairly broad; that is,
7 the authority to require additional information, and
8 even tests that are not included in 40 CFR, Part 158
9 have been required upon occasion; special testing that
10 might be appropriate to particular chemicals.

11 Now, as I mentioned and as is indicated
12 on this overhead, some tests are done on a formulated
13 product, some tests are done on the active ingredient
14 alone. As a general rule of thumb, I would say that
15 the tests that are done to determine the inherent
16 chemical and toxicological characteristics of the
17 active ingredient are done on the active ingredient and
18 in that area I would put the toxicology tests and some
19 of the chemical tests. However, there are some
20 wildlife toxicity tests that are also done on active
21 ingredient and so on.

22 Generally, the tests that are trying to
23 determine how the chemical will behave in the
24 environment and the potential for human or non-target
25 organism effects under actual conditions of use, those

1 tests are done on a formulated product as formulated
2 for use, so that the results give as accurate a picture
3 as possible of what the impacts of actual use of that
4 product are going to be.

5 Now, as you know from previous testimony,
6 the ingredients in a pesticide that are not active
7 ingredients, that do not of themselves have pesticidal
8 properties are called inert ingredients. What you
9 should know is that in the United States inert
10 ingredients, any ingredients in a product that is
11 applied to growing food crops must either receive a
12 tolerance, what is called in Canada a maximum residue
13 limit, or must be specifically exempted from the
14 requirement for such tolerance by the agency and this
15 applies a review of safety information.

16 Chemicals that have met that criterion;
17 that is, that have exempted from the requirement for
18 tolerance are published in the Code of Federal
19 Regulations at 40 CFR, Part 1(a).1001, one thousand and
20 one. To the best of my knowledge, that list is the
21 reference that manufacturers use when selecting
22 ingredients to use in formulations for new products;
23 not just not food use products, but all products and
24 that is the best of my understanding.

25 Now, you are also aware from previous

1 evidence that in 1987 the EPA formulated a policy for
2 regulating inert ingredient and I believe a copy of
3 that policy had been submitted to you previously as
4 Exhibit 725.

5 In developing this policy, the EPA
6 promulgated four lists of inert ingredients, and I
7 would like to explain to you a little bit, to the best
8 of my knowledge, how those lists were generated. What
9 EPA did was to go back into its historical records of
10 inert ingredients that were present in registered
11 products up until that time and they did a complete
12 listing of all of those inerts.

13 Now, you should know that that database
14 contains a lot of entries that are no longer accurate.
15 For one reason or another, people sometimes fail to
16 update -- sorry, people fail to remove from the EPA
17 files confidential statements of formula that are no
18 longer applicable to registered products. They have an
19 obligation to file a new one. They don't also take the
20 administrative step of saying: EPA, please pull the
21 old one, get rid of it.

22 The other thing is that people --
23 registrants, for whatever reason, tend to keep
24 registrations active, even for products that aren't
25 really being sold. So in going through that database,

1 EPA must have come up with inert ingredients that
2 really are no larger in use for one reason or another.
3 The reason I know this is because Environ Corporation
4 provided support to EPA in the development of that
5 inert ingredient policy and one of things we did was to
6 help them discover, shall I say, the strengths and
7 limitations of their own database for coming up with
8 this list of inert ingredients.

9 So once they had established this
10 universe of inert ingredients, they proceeded to do a
11 search of the scientific literature to determine
12 whether or not any of these inserts had been identified
13 as having toxicological effects. As you know, from
14 Exhibit 725, List 1 contains about 55 ingredients that
15 have already been demonstrated in some form to have
16 some sort of effect and these effects range from a
17 variety of human health effects to bioaccumulation and
18 certain environmental effects.

19 In putting this list together, EPA did
20 not -- in the exhibit that you have, EPA did not
21 designate the reason for listing any one of those
22 chemicals. Those reasons are available but in a
23 different document.

24 Now, List 2 consists of inert ingredients
25 that have a high priority for testing. The reason they

1 have a high priority for testing is because some
2 scientific concerns have been raised about potential
3 toxicity or environmental effects. Many, if not most
4 of the chemicals on List 2, have already been
5 identified by the National Toxicology Program as
6 canidates for testing.

7 List 4 is the list that contains
8 ingredients that are of no concern. These ingredients
9 are generally recognized as safe or grass status - that
10 is conferred by the U.S. Food and Drug Administration -
11 or they have been through some sort of review or
12 they're exempted from the requirement of the tolerance,
13 but in general there are no concerns about the list for
14 chemicals.

15 And then there is List 3 which is
16 essentially the other category, things that have not
17 yes been categorized. This regulatory policy is
18 intended to be fluid, as more information comes in
19 chemicals may move from one list to another. It is
20 fully expected that chemicals that are now on List 2
21 will end up either on List 1 or List 4 as testing
22 proceeds.

23 Now, for chemicals that are -- for inert
24 ingredients that are on List 1, the EPA has established
25 data requirements. There will be no new registrations,

1 new products registered containing those inerts unless
2 people supply data to show that the presence of those
3 inerts doesn't pose a hazard and there is an extensive
4 list of data requirements that is being posed.

5 For existing products, existing
6 registrations that contain List 1 inerts, the agency
7 required that the labelling of products containing
8 those inerts carry a warning statement as of October
9 1987 and that warning statement has to say something to
10 the effect of: Warning, this product contains the
11 toxic inert so and so.

12 Now, the requirement to put that kind of
13 a statement on a label is a strong incentive to the
14 manufacturer to get that inert ingredient out of the
15 formulation and that's for two reasons. First of all,
16 because, as you know, in the United States the identity
17 of inert ingredients is confidential business
18 information, it is trade secret, the formula is a
19 secret and the manufacturer doesn't like to divulge
20 that.

21 The second reason is that the appearance
22 of a warning statement like that on a label quite
23 rightly alarms consumers, purchasers, the users of the
24 product and has negative impacts on sales, and EPA knew
25 what it was doing when it imposed that labelling

1 requirement. The intent behind it was to get people to
2 get those List 1 ingredients out of their formulations
3 and to substitute things that were less toxic.

4 I won't go through the details of that
5 exhibit since you already have it before you, but
6 testing requirements have also been established for the
7 List 2 ingredients. They will be dealt with on a
8 case-by-case basis as they become before the agency
9 and, as I said, this entire process will be ongoing.
10 So as new information comes in, things will continue to
11 change.

12 Q. I believe we are ready to move to the
13 next overhead?

14 A. Yes. The next overhead, please.

15 MR. CASSIDY: That will be page 3 of
16 Exhibit 1240, Madam Chair.

17 DR. RACHMAN: Now, as you know from
18 previous testimony, I believe Dr. Ritter talked about
19 this in detail, the United States EPA has specific
20 protocols and guidelines established for the conduct
21 and the reporting of the studies that are required for
22 registration and these things determine whether or not
23 the data that are developed are acceptable for
24 reviewing the registration process.

25 The guidelines contain several parts.

1 First of all, they are referred to as EPA pesticide
2 assessment guidelines and you will hear people
3 abbreviate that as the guidelines. The first part of
4 the guidelines consist of the standards and the
5 protocols for the individual tests that are outlined in
6 40 CFR, Part 158. Those protocols establish things
7 like the number of animals that should be used, how the
8 dosage should be selected and administered, how the
9 animals should be housed and those sorts of things.

10 In recent years, EPA has issued addenda
11 to those guidelines which specify not only how the
12 tests should be done, but how the results should be
13 recorded. So that the test report itself has to be
14 organized in a very specific manner, sections have to
15 appear in a certain order, they have to be explained in
16 a certain way and so on.

17 Let's skip No. 3 here for a moment, I
18 will come back to it. The last item, good laboratory
19 practices. These are regulations that cover the way a
20 test is performed and how the data are handled. These
21 have applied to toxicology studies since 1978. Since
22 October 1989, these regulations now apply to all
23 testing done in any subject area for pesticide
24 registration in the United States. These good
25 laboratory practices or GLP requirements can be thought

1 of as kind of an institutionalization of standard good
2 scientific practice. These prescribe procedures that
3 any good scientific investigator working in a reputable
4 laboratory would normally use in designing and
5 executing a test; however, there are a few requirements
6 that sort of go above and beyond what a good scientist
7 would formally do and those requirements are in the
8 areas of record keeping and retention, sample retention
9 after the test is complete, those sorts of things.

10 So what I am getting at here is that the
11 fact that a test does not meet GLP standards is not
12 necessarily an indication that it is not a
13 scientifically valid test. It could be failing to meet
14 some of those requirements for sample retention or
15 record keeping that are really way beyond what a good
16 scientist would normally do.

17 Now, a failure to meet these criterion
18 constitutes a data gap; that is, if a test is not done
19 according to a standard accepted protocol or if it is
20 not reported according to the standard report formats
21 or if it was not performed according to the letter of
22 the GLP regulations, that test is not acceptable for
23 review by the agency. The agency won't even look at
24 the scientific content of the study if it doesn't get
25 past that first stage of review.

1 Point No. 3 here, the EPA standard
2 evaluation procedures, are not properly part of the
3 guidelines and perhaps I should not have put them on
4 this slide. Those are actually standard operating
5 procedures of EPA's that determine how an EPA reviewer
6 should review particular studies. The reason I
7 included it here is because potential registrants look
8 at those SOPs, they contain very important information
9 about what EPA is looking for in a study, and so it
10 provides information to registrants when trying to
11 design studies.

12 Okay. Now, as I said, data gap applies
13 to cases in which a study does not meet these criteria.
14 There is also another definition of data gap and that
15 is a situation in which information is missing. That
16 could arise for several reasons that I could think of.
17 Commonly it comes up because the data requirement could
18 not have been anticipated by the registrant at the time
19 that the pesticide was registered.

20 In the registration process, it often
21 happens that EPA requires some additional special study
22 that is not on the Part 158 list or which may be
23 indicated as conditional and the registrant is not
24 certain that that will be a requirement. In the
25 initial review of that package, the EPA may designate

1 that as a data gap. If it's not a serious data gap and
2 the absence of that does not detail a significant
3 adverse effect, EPA may go ahead and grant what's
4 called a conditional registration. That means that the
5 registration is granted, conditional upon the
6 registrant providing that additional information at a
7 certain point in the future.

8 If the agency makes the determination
9 that the absence of that data constitutes a potential
10 for a significant adverse effect, they will not grant
11 the conditional registration, they will wait until
12 those data are supplied and they have a chance to
13 evaluate them.

14 Now, I would like to point out some
15 additional things. Let's go to overhead No. 4, please.

16 These are other requirements that apply
17 to data developed for registration. I did not include
18 this in the statement of evidence, but I would like to
19 bring this to your attention. The agency has
20 established what they call flagging criteria. The
21 intention behind this is to prioritize for agency
22 attention certain information that may indicate a
23 potential for significant adverse effects.

24 These requirements apply to any new
25 information that is generated and submitted to EPA,

1 either in connection with a new registration or the
2 support of an old registration, an update of the
3 database. What these regulations require is that if
4 the results of the studies at issue contain certain
5 factors, certain results obtained, you must put a
6 statement on the cover bringing to the agency's
7 attention that dose effects have been noted and what
8 that does is literally raise a red flag. When the
9 agency receives that package those studies go into a
10 priority review. The whole system is intended to allow
11 the EPA to be extremely responsive to new information
12 coming in that may have any significant -- that may
13 have the potential to indicate adverse effects.

14 These criteria apply to toxicology data
15 and they apply to all the major areas of testing,
16 oncogenicity or the cancer studies. I might point out
17 that EPA uses the oncogenicity because they are
18 concerned with not only malignant tumors, but also
19 benign tumors.

20 The chronic feeding studies and
21 subchronic feeding studies, I believe you know about
22 those, those are the two year or 90-day studies.
23 Teratogenicity is birth defects. Neurotoxicity and
24 reproduction I think are self explanatory.

25 If we can have the next overhead, please.

1 MR. CASSIDY: Q. I understand that this
2 section of your evidence, Dr. Rachman, will deal with
3 the post registration or requirements imposed by the
4 EPA?

5 DR. RACHMAN: A. That's correct. Once
6 registration is obtained the registrant is not
7 finished; there are more obligations that apply.

8 There is a section in FIFRA in the law,
9 section 6(a)(2), that specifies that any time
10 information comes to a registrant on a registered
11 product and that information has the potential to
12 indicate adverse effects, the registrant must make EPA
13 aware of that information within 15 working days of
14 finding out about it.

15 Now, there are regulations that have been
16 developed that go into great detail about what kind of
17 information must be recorded and what I have done on
18 this overhead is to just summarize in a very general
19 way the types of information that are covered by this
20 requirement.

21 MR. CASSIDY: And, Madam Chair, this
22 evidence will relate to a issue raised in the scoping
23 session by yourself dealing with the EPA registration
24 process and its ability to make changes as scientific
25 information becomes available.

1 Q. Go, ahead Dr. Rachman.

2 DR. RACHMAN: A. The registration
3 relates to completed toxicity studies and they also
4 cover certain incomplete toxicology studies. If you
5 have a study that's ongoing and during the course of
6 that study you notice certain things happening to the
7 animals and test organisms that may signal the
8 potential for adverse effects, you have to report that
9 to the agency even before the test is completed, even
10 before you have all the information in that allows you
11 to determine whether or not that really is an adverse
12 result.

13 Epidemiological studies are covered,
14 including studies of efficacy of product. If a
15 registrant begins to get information that his product
16 is no longer effective for the purposes for which it
17 was registered, that is a reportable incident. If
18 residues in the diet or in the environment are found to
19 exceed the levels that were anticipated during the
20 registration process based on the registration data,
21 that has to be reported to the agency. Also any
22 incident that occurs, poisoning, et cetera that come to
23 the registrant's attention. All that information has
24 to be reported.

25 MADAM CHAIR: Excuse me, Mr. Rachman,

1 does that include -- does that focus more on the
2 information that the registrant will obtain through the
3 review of scientific literature as opposed to its own
4 testing that it has done on its product?

5 DR. RACHMAN: It includes --

6 MADAM CHAIR: That's a wider net for a --

7 DR. RACHMAN: That is exactly the term I
8 was going to use. The regulation casts a very wide
9 net, is doesn't restrict the registrant to look at one
10 particular body of information, so it does include the
11 tests that's are underway for whatever reason, the
12 scientific literature, case reports from the medical
13 literature, reports from various state agencies that
14 monitor pest use and accidents and so on.

15 I can conceive of cases, for example,
16 where there might be a report in the media of an
17 incident involving a pesticide use and my
18 interpretation of the regulations would be that the
19 registrant would have the responsibility to follow-up
20 on that and determine whether or not that was a
21 reportable result under Section 6(a)(2).

22 MADAM CHAIR: And how long does that
23 obligation continue for the registrant?

24 DR. RACHMAN: Forever once the product is
25 registered.

1 MADAM CHAIR: Forever.

2 DR. RACHMAN: Yes. And compliance --
3 there are 15 work days for compliance once the
4 information -- when the registrant becomes aware of the
5 information and there are substantial fines for
6 non-compliance.

7 Another very important aspect of this
8 whole procedure is that when the agency receives these
9 class disclosures they are made public and reports of
10 them appear in the Trade Press routinely. By the Trade
11 Press I mean publication of pesticide and toxic
12 chemical use --

13 Q. I'm sorry?

14 A. Toxic chemical use published in the
15 States. And so this is a very rapid technique for
16 making the public aware of adverse effect disclosures
17 that have been filed.

18 I think I would like to go on and talk
19 about reregistration.

20 MR. CASSIDY: That overhead is No. 6,
21 Madam Chair, of Exhibit 1240.

22 Q. I would ask you, Dr. Rachman, to
23 describe first the history and authorization of the
24 registration process and then the process itself.

25 DR. RACHMAN: A. I'd just like to return

1 to the adverse effects for one moment, I neglected to
2 make an important point which was implicit I think; and
3 that is, the agency has the authority to take
4 regulatory action at any time based upon that 6(a)(2)
5 information that is submitted.

6 Q. What type of regulatory action are
7 you referring to, Dr. Rachman?

8 A. Well, I will talk a little bit later
9 about the remedies that are available to the agency in
10 circumstances where risk is presumed.

11 MR. CASSIDY: And that will also relate
12 to the scoping session issue raised by yourself, Madam
13 Chair.

14 Q. If you could then move to the
15 reregistration process which is described in the
16 evidence and in the overhead No. 6, Exhibit 1240,
17 please?

18 DR. RACHMAN: A. Yes. Now,
19 reregistration has been required since 1972 when FIFRA
20 was amended to incorporate that requirement and, in
21 fact, in 1988 the law was amended and there is a whole
22 new section of FIFRA now, Section 4, which deals
23 extensively with reregistration requirements and
24 procedures.

25 I would like to point out that I made an

1 error in my statement of evidence. I referred to FIFRA
2 Section 3(g), this is on page 11 of my statement of
3 evidence -- I referred to Section 3(g) of FIFRA, that
4 was the old FIFRA. I should have put Section 4 because
5 as of 1988 all of the registration requirements, I
6 believe, have been moved to Section 4.

7 The 1988 amendments formalize the
8 reregistration process, set out procedured and time
9 lines. Very important. A nine-year deadline has been
10 imposed by which time EPA must complete the
11 reregistration of all active ingredients and they have
12 been directed to prioritize this reregistration process
13 based on the same sorts of potential exposure criteria
14 that I mentioned before.

15 They were required to establish four
16 lists of chemicals and associated deadlines for the
17 submission of data and review of those data and so on.
18 Those lists have been now been published. List A
19 consists of active ingredients for which a registration
20 standard has already been issued. Those chemcials are
21 already in the process somewhere. There are
22 approximately 192 of them.

23 MADAM CHAIR: Excuse me. Is this
24 separate from the four lists we just discussed?

25 DR. RACHMAN: Yes, Madam Chair. Those

1 lists refer to inert ingredients, reregistration of
2 active ingredients, although I should point out that in
3 reviewing the data on active ingredients and preparing
4 the registration Standard, EPA also considers the
5 formulated product. It is just that administratively
6 it is all designated by the name of the active
7 ingredient.

8 Let's see. So List A consists of the
9 chemicals that -- they are already in the works. Lists
10 B, C and D are the rest of them and the deadline for
11 submitting the first information on List B chemicals
12 has just passed, the deadline for the first information
13 submission on List C chemicals is in July, and I
14 believe the deadline for the List D chemicals is three
15 months after that. So things are going to proceed at a
16 very rapid clip. Nine years is not a long time, I
17 should point out, for doing this kind of extensive
18 review and I will get into that a little bit later.

19 The Congress directed EPA to prioritize
20 these active ingredients for reregistration
21 consideration using the following factors: whether or
22 not they are used on food; whether or not they have
23 significant outstanding data gaps; and whether or not
24 there is significant potential for worker exposure.

25 So the chemicals that have the highest

1 ranking according to those three criteria are on List
2 B, so they were the first ones to start the process;
3 the next lower set of rankings is List C; and then the
4 ones with the least concern in that area are in the
5 List Ds.

6 Now, on this overhead, what I'm trying to
7 provide to you is my own personal understanding of this
8 process and how it works. There is at present,
9 interestingly enough, no definition of reregistration
10 and that's because, by its very nature, it is an
11 ongoing cyclical process. I don't believe it was ever
12 intended to have an ending

13 Reregistration was mandated in the first
14 place because of the recognition that scientific
15 advances eventually mean that data requirements change
16 and protocol requirements change and as science
17 continues to advance, data requirements will change to
18 change. I am sure that years from now we will be
19 testing pesticides for effects that we never even
20 dreamed of testing for, simply because basic research
21 will lead us in the direction of evaluating the
22 concerns.

23 So what typically happens in the
24 registration standard process is that EPA starts by
25 taking a look at what's in the file, what's in the

1 database for the chemicals that's been registered and
2 determining what data should be supplied to fulfill the
3 registration requirements.

4 Now, that includes two sorts of
5 considerations. One, are there no new data
6 requirements today that did not apply when the chemical
7 was first registered; and, secondly, are there any new
8 protocols in place today, new guidelines that were not
9 in place when the chemical was first registered.

10 A deficiency in either of those areas;
11 that is, either missing information or information that
12 doesn't come up to the grade are current contemporary
13 protocols, either deficiency is labelled a data gap in
14 the registration standard process and you can't really
15 tell from looking at the EPA documents what kind of
16 data gap you are looking at.

17 You can appreciate, I am sure, that there
18 is a vast difference in the quality of those two kinds
19 of data gaps. In the one case, you have absolutely no
20 information available to you as to whether or not you
21 have got a potential adverse effect in the area that
22 you are talking; in the other case, you do have
23 information, but what you've got for some reason does
24 not up come to current EPA standards.

25 Now, in fact there are some good examples

1 of that with some of the chemicals of interest to this
2 proceeding, 2,4-D, for example. Dr. Ritter talked in
3 his testimony about data that was available to Canada,
4 that was not available to the EPA in the areas of -- I
5 believe it was mutagenicity and metabolism.

6 The EPA said that all of those studies
7 were unacceptable. They were unacceptable because they
8 didn't meet current EPA guidelines for the performance
9 of those studies, and yet the data are of sufficient
10 scientific quality that the expert panel conveyed by
11 the Ministry of the Environment here in Ontario relied
12 on them in assessing potential risks of 2,4-D.

13 The way the EPA process works is that if
14 the data don't pass that initial screen as to form, the
15 scientific content is not even evaluated, they don't
16 even look at it, and they designate in the registration
17 standard that the information is not available, there
18 is a data gap.

19 Now, the EPA will issue a document that's
20 called the registration standard, I have been referring
21 to that. That's a document that lays out what the data
22 requirements for the active ingredient should be -- I
23 should say are, what the data requirements are, whether
24 or not the EPA currently has information that satisfies
25 those data gaps and -- I'm sorry, that satisfies those

1 data requirements and where data gaps are indicated
2 deadlines, by which time new information must be
3 submitted if the registration is to continue.

4 In going through all the existing data,
5 the EPA evaluates the potential risks and if the data
6 that are in the file, even if they are incomplete, if
7 they should indicate that there exists a potential
8 adverse effect, then the EPA has remedies available to
9 it and it will often take interim action at that point,
10 and I will talk about that in a moment.

11 When the new information comes in, and it
12 may take a couple of years, depending on what kinds of
13 tests are required and how long they take to run, there
14 is what's called a second round review. The EPA
15 re-evaluates the risk picture in light of new evidence,
16 issues new findings as to the potential risk and
17 sometimes requires more data.

18 Now, because it can take as much as four
19 years for some of these tests to be done, particularly
20 the chronic studies, and it can take a year or two for
21 EPA to review the studies, in the meantime, as the
22 guidelines for other studies have been updated, new
23 data gaps can spring up. So you can see that this is
24 as cyclical process. While you are filling one data
25 gap another one may pop up, so then you have to go back

1 and review that study and that's just the way it was
2 intendewd to be, this constant upgrading of the
3 database.

4 This is presenting some interesting
5 problems for EPA now in light of this nine-year
6 deadline that's been imposed by the 1988 amendments to
7 FIFRA. A nine-year deadline implies that at the end of
8 nine years they have to be at the end of something and
9 they are right now wrestling with a definition, at what
10 point are they going to be able to say to Congress that
11 they have complied with that nine-year deadline when in
12 fact they have a process that was intended to go on
13 forever, and this should be very interesting to watch.
14 There are high level meetings going on in Washington
15 for the last couple of months trying to wrestle with
16 this very important policy problem.

17 Let me just remind you that in the
18 development of this new information, anything that's
19 required under a registration standard, those flagging
20 criteria apply. So that if you are doing a new toxic
21 study and you get a result that's indicated you have to
22 bring it to the agency's attention immediately.

23 The 6(a)(2) adverse effects disclosures
24 also apply because you are dealing with a product
25 that's already registered. So if you are doing a new

1 cancer study, for example, and you get some adverse
2 effect that indicate a different order of toxicity than
3 your old studies indicated, you may have to disclose
4 that under the adverse effects disclosure.

5 Again, this illustrates that EPA is --
6 EPA procedures are set up so that they are responsive
7 to incoming information that indicates potential for
8 adverse effects.

9 I would just like to remind you that the
10 existence of a data gap does not imply a risk of any
11 kind. I can't emphasize this point enough. Because of
12 the different kinds of deficiencies that are all
13 covered by that same term, 'data gap', you have to be
14 very careful. If someone says to you there is a data
15 gap on a chemical, it does not necessarily imply that
16 there isn't any information there that you can use to
17 evaluate whether or not there is a risk and it doesn't
18 imply that evaluation could not be done. What it means
19 is that the information may not meet EPA's criteria.

20 The question you should ask always, I
21 would suggest, is whether or not there there is any
22 information there at all that's useful in a scientific
23 sense, whether the information is missing. Whether or
24 not it is acceptable to EPA has no bearing practically
25 on its scientific validity.

1 Q. All right. I understand we are ready
2 to move to the next overhead.

3 A. I believe we are.

4 Q. And this will be overhead No. 7 of
5 Exhibit 1240.

6 Madam Chair, this section will be -- I
7 would like to ask Dr. Rachman to describe the EPA's
8 authority to act when significant risks are identified
9 and she will deal with the powers of the EPA once that
10 occurs.

11 DR. RACHMAN: Okay. I have tried to
12 indicate how information that suggests potential
13 adverse effects can come to EPA in a variety of ways
14 and at any time, and now I would like to talk to you
15 about some of the remedies that they have available to
16 them when they decide there is a problem or potential
17 problem.

18 I have tried to organize these, just for
19 discussion purposes, in ten -- in order of, shall we
20 say, ease of EPA imposing this action. The amendment
21 of terms of registration, for example, the first one is
22 something that EPA can do at any time under the
23 authority of FIFRA. Whenever they make the
24 determination that there is some risk they can impose
25 conditions on the registration; that is, changes on the

1 labelling, for example, requirements for protective
2 clothing, something like that, that are intended to
3 mitigate the risk and they routinely do this during the
4 registration standard process while they are waiting
5 for more information to come in that will allow them to
6 do a more in-depth risk assessment.

7 For example, some of the chemicals at
8 issue here are involved in the registration standard
9 process. I can give you some examples. Now, let me
10 point out that these risk reduction measures are
11 specific to individual uses of the chemical, okay. The
12 examples I am going to give you are not for forestry
13 uses of these chemicals, although these are the
14 chemicals that are approved for forestry use in
15 Ontario.

16 The reason I will not give you examples
17 for forestry uses is that we were unable to find any
18 cases in which EPA has imposed risk reduction measures
19 on forestry uses. There have been no risks identified
20 specific to forestry uses; that is, human health risks
21 and there have been no interim human health risk
22 reduction measures imposed for forestry uses.

23 Now, with that said let me give you some
24 examples. The fenitrothion registration standard,
25 which was issued in 1988, imposed an interim 24 hour

1 re-enty interval for greenhouse uses of fenitrothion.
2 This is because EPA felt that there was not enough
3 information available in the files about the potential
4 exposures of greenhouse workers using fenitrothion. So
5 they imposed a data requirement on the registrant to do
6 such a study and in the meantime, until those data are
7 submitted and evaluated, there is a 24 re-enty interval
8 which means that after a greenhouse is treated with
9 fenitrothion workers cannot re-enter the premises for
10 24 hours and signs have to be posted and so on.

11 I can also give you another example for
12 glyphosate. Again, not a forestry related use. This
13 one applies, I believe, to the agricultural and aquatic
14 uses of glyphosate. In developing a registration
15 standard, the EPA became aware of some incident reports
16 from the State of California - California has a
17 monitoring system in place - and apparently there were
18 some reports of irritation and severe irritation,
19 ocular and dermal, I believe, from uses of glyphosate
20 by mixers and handlers and these incidents splashing of
21 the formulated product and so on. So in this case, EPA
22 imposed a label requirement for protective clothing of
23 specifically face shields and so on to cut down on the
24 probability of those kinds of worker exposures and
25 accidents.

1 I will review for you at the end of my
2 talk the EPA status of the chemicals at issue here in
3 this proceeding and I will come back to the interim
4 risk reduction measures at that time and make
5 corrections to overhead No. 11 in your packet.

6 Okay. The next remedy available to EPA
7 is special review, and I guess we could go to the next
8 overhead at this point.

9 Q. All right. That will be overhead
10 No.8 of Exhibit 1240.

11 A. Overhead No. 8. The process that's
12 now called special review was formally called
13 rebuttable presumption against registration or RPAR,
14 and thankfully EPA changed the name. RPAR was
15 originally intended to evaluate the evidence to see
16 whether unreasonable adverse effects are likely to
17 occur. This is the procedural review of the evidence
18 to make that determination.

19 The original RPAR did not specify that
20 EPA consider exposure data and the result I think was a
21 very cumbersome process because every time there was
22 some indication of inherent toxicity of a compound they
23 would have to do this indepth evaluation.

24 As you know from previous evidence, Dr.
25 Ritter talked about this in detail, there is no risk if

1 there is no exposure. There may be toxicity, but if no
2 one is exposed there is no risk. So in 1985 -- well,
3 in 1978 FIFRA was amended to take this consideration
4 into account, and in 1985 EPA formalized the new
5 process and gave it the name special review to signify
6 that there had been a change.

7 There are specific criteria for the
8 initiation of a special review and the criteria
9 specified that the process can only be initiated if EPA
10 has validated test evidence or other significant
11 evidence raising prudent concerns of unreasonable
12 adverse risk, and the word risk is in the regulations,
13 and of course that implies not only toxicity but also
14 exposure.

15 What the special review process is, then,
16 is a more indepth review of the evidence. If the
17 agency makes a finding that there is evidence
18 indicating a risk, they will begin the process of an
19 indepth determination of whether unreasonable adverse
20 effects occur and, as I showed on an earlier slide,
21 unreasonable adverse effects includes not only risk,
22 but also benefit. So you can think of the special
23 review process as an indepth risk benefit evaluation of
24 the use of the pesticide.

25 Now, this slide here, No. 8, shows you

1 the initiation criteria in the area of human health
2 effects. There are also criteria for environmental
3 problems and I have the not dealt with those since our
4 evidence is dealing with human health effects.

5 Again, the standard of the evidence is
6 validated test or other significant evidence. I would
7 point out that EPA has to include not only the active
8 ingredient in its consideration, but also impurities,
9 metabolites, and so on. The criteria you can see here
10 are very broad, a wide range of effects could indicate
11 a concern provided that the evidence is of sufficient
12 quality the agency can initiate a special review.

13 Special reviews are rare and the reason
14 that they are rare is because the agency has to have a
15 high quality of evidence in order for be able to
16 initiate one. They have to be able to make the
17 determination that there is the potential for
18 unreasonable adverse effect for a risk, and frequently
19 the evidence that's available just does not allow them
20 to make that determination. We have provided in our
21 statement of evidence EPA's latest position with
22 respect to 2,4-D.

23 Q. And I understand you wish to update
24 that with respect to what has happened since?

25 A. Yes, I will do that. Can I wait

1 until we get to slide No. 11?

2 Q. Certainly.

3 A. I should say that this is not the
4 latest position. This was the latest position at the
5 time we prepared our statement of evidence. I will
6 update you at the end of our talk.

7 Q. Yes.

8 A. In this notice, EPA announces that it
9 has decided not initiate the special review on 2,4-D,
10 and to paraphrase liberally, the reason is that the
11 evidence just does not indicate that there is a risk of
12 carcinogenicity.

13 Q. All right. The next overhead?

14 A. Let me just check here for a moment.
15 I would just like to say a word about the procedures of
16 special review because some of those terms may come up.
17 Yes, let's go to overhead No. 9.

18 Q. This is overhead No. 9 in Exhibit
19 1240.

20 A. The EPA special review process is
21 like the registration standard process, lengthy.
22 Probably it would be fair to say that a special review
23 cannot be concluded in under two years except under
24 what I would say unusual circumstances. The
25 presumption starting a special review is usually that

1 there is a risk that has been identified that may be so
2 serious that the registration has to be cancelled.
3 That's usually the story, and then the registrant has
4 the burden of providing evidence to EPA to show that
5 the risk not that great.

6 EPA usually imposes data requirements and
7 asks for specific information that it would like to see
8 in order to do a better determination of the potential
9 risk.

10 MADAM CHAIR: Excuse me. Is that after
11 they cancel the registration or--

12 DR. RACHMAN: No, it is not.

13 MADAM CHAIR: --it would follow the
14 cancellation until they...

15 DR. RACHMAN: Yes. They may make an
16 initial proposal that cancellation might be required,
17 but they can't proceed to cancellation until they have
18 made the final determination that there does exist an
19 unreasonable adverse effect and that there is no way to
20 mitigate it except to cancel the registration.

21 MADAM CHAIR: And they can't do that
22 usually under two years?

23 DR. RACHMAN: That's my impression, Madam
24 Chair. I have not done a thorough study of all the
25 special reviews that have been done, but I have been

1 involved with a couple on specific chemicals and my
2 recollection is that the procedure took in the order of
3 two to three years to come to its regulatory
4 resolution, which was not always cancellation, and I
5 will talk about that.

6 MADAM CHAIR: And in the meantime in that
7 two year period when possible cancellation and the
8 reason for it is being studied, does EPA put on special
9 conditions to mitigate that as well?

10 DR. RACHMAN: They may do so, yes,
11 exactly. The initiation notice kind of puts the
12 registrant on notice that EPA has identified a certain
13 potential risk and kind of, you know, casts the net for
14 information and then also advises the registrant of
15 particular data requirements and deadlines for
16 supplying the data.

17 The position documents, there have been
18 as many as four that I am aware of, sequential
19 documents, within the course of a special review. Each
20 one is an update of the agency's position vis-a-vis the
21 risk and the proposed regulatory measures in light of
22 evidence that has come in since the last position
23 document.

24 This is a very important point, that the
25 standard of evidence here in a special review

1 proceeding is very high and so the agency cannot move
2 quickly through this process. These are generally very
3 difficult scientific questions that are dealt with and
4 there is a lot of interpretation, a lot of
5 scientific -- back and forth and until there is a
6 resolution and a final risk assessment that people are
7 happy with, it takes some time.

8 Now, the outcome -- let me just mention
9 the scientific reviews which is point No. 3. Section
10 25(d) of FIFRA directs that whenever EPA is proposing
11 the cancellation of a registration they have to submit
12 that proposal and the scientific rationale to a
13 scientific advisory panel, and the section goes on to
14 give criteria for selection of panel members and so on.

15 Basically these are outside independent
16 scientists with recognized credentials and a lot of
17 experience and they serve limited terms on the panel
18 and they advise the agency about proposed
19 cancellations. The agency also makes liberal use of
20 the SAP to evaluate proposed regulatory policies and
21 other scientific issues that arise in the course of
22 registration. They meet regularly.

23 So this outside peer review is a part of
24 this process and frequently the SAP will review an
25 agency proposal and say that they don't agree with the

1 agency's position, the evidence doesn't support it,
2 they need more information and so on, and that
3 frequently is one reason why the process takes so long,
4 because these scientists direct that other things
5 should be considered, other data should be developed
6 and so on. The SAP role is advisory; EPA does not have
7 to listen to what they say, but in practice its
8 suggestions are generally taken into account.

9 Now, the outcome of a special review is
10 not a regulatory action, it is a recommendation for an
11 action. The outcome of a special review is that
12 weighing of risk versus benefit for a particular use,
13 the use that's at issue or for all uses, depending on
14 what the problem that is identified is. If that
15 risk/benefit relationship cannot be brought into
16 acceptable balancing, then cancellation is an option.

17 Could we go to the next overhead, please.

18 Q. Overhead No. 10, Exhibit 1240.

19 A. These are the two regulatory options
20 that EPA has available once a significant risk has been
21 identified, the final determination of risk has been
22 made. Cancellation requires, as I've said,
23 unreasonable adverse effects have been identified and
24 the project -- I'm sorry, the product does not meet the
25 registration requirements that I illustrated on my

1 first slide.

2 Now, the thing about cancellation is that
3 the law and regulations provide that effected parties
4 may ask for a hearing which, of course, they almost
5 always do and those hearings are quite lengthy and the
6 cancellation process can, therefore, take in the order
7 of a couple of years.

8 MADAM CHAIR: Excuse me. A hearing
9 before whom?

10 DR. RACHMAN: It is an administrative
11 hearing.

12 MADAM CHAIR: Before --

13 DR. RACHMAN: Before an administrative
14 law judge on the question of unreasonable adverse
15 effects and agency procedures in evaluating those
16 risks.

17 While this is going on or even during a
18 special review, registrants may choose to voluntarily
19 cancel their own registrations. You will occassionally
20 hear of this happening. Frequently -- well, I
21 shouldn't say frequently. My impression of why this
22 happens is that in some cases the registrant may
23 recognize that there is in fact a significant risk and
24 that there is no point in going through a cancellation
25 proceeding because ultimately the product will be

1 cancelled anyway.

2 There is another situation that occurs in
3 the United States and that is, because all of these
4 proceedings were public and there is a tremendous
5 public concern about pesticides, as there is here in
6 Canada, adverse publicity or publicity about adverse
7 effects has a deleterious effect on sales. So in some
8 cases manufacturers may just make the decision that
9 selling the product is no longer feasible, but for
10 whatever reason, it is possible for a manufacturer to
11 voluntary cancel at which point the process just ends.

12 If there is a special review going on,
13 EPA simply publishes a notice terminating the special
14 review. If there is a cancellation proceeding in place
15 they just terminate that and that's it, cancellation is
16 effective.

17 Okay. So the cancellation proceeding
18 takes some time. Now, in going into a cancellation
19 proceeding, EPA will do an analysis of how much risk,
20 if you will, will attain during the period it's going
21 to take to cancel this product and if they decide that
22 that's an unacceptable risk, let's say two more years
23 of use of this product or 18 months or whatever is
24 going to cause an unacceptable risk, instead of
25 cancellation they have the option of suspension.

1 In order to propose suspension they have
2 to find -- make a finding of imminent hazard. Imminent
3 hazard is what I just explained. It's a finding that
4 during the time it would take to cancel this product
5 the risk would be unacceptable.

6 Now, registrants have the option of
7 requesting a hearing on suspensions as well, even
8 though suspensions are effective within 30 days of the
9 notice. If the registrant requests a hearing, they
10 have to have a hearing and that may stretch the
11 proceeding out to several months.

12 If EPA decides that during that period of
13 several months, the time needed for a suspension
14 hearing, the risk would be unacceptable, they have a
15 further option, they can do an emergency cancellation
16 which is -- I'm sorry, an emergency suspension which is
17 effective immediately. No hearing.

18 This is a very, very rarely exercised
19 option, but in fact it has been exercised recently and
20 that was the emergency suspension of dinoseb. Dinoseb
21 is not used for forestry. A registration standard was
22 issued on dinoseb in 1984, it has a variety of
23 agricultural uses. As a result of that registration
24 standard, the registrant had to do a new teratology
25 study, a birth defect study, and when the results of

1 that study were submitted to EPA, the EPA determined
2 that the margin of safety for handlers and applicators,
3 mixer, loaders, and so on, people handling dinoseb, the
4 margin of safety was unacceptable.

5 Because of where they were during the
6 calendar year, the dinoseb use season was upon them and
7 the agency made the determination that the number of
8 people that would be at risk from the use of dinoseb
9 within the coming six months was unacceptable and under
10 those conditions they moved for an emergency
11 cancellation of dinoseb which became effective -- I
12 think it was 1985. I would have to check that for you.

13 MR. CASSIDY: I think we are ready to
14 move on to the next section and this section of Dr.
15 Rachman's evidence, Madam Chair, deals with the EPA
16 status of pesticides approved for forestry use --

17 MADAM CHAIR: Excuse me, Mr. Cassidy.

18 MR. CASSIDY: Yes.

19 MADAM CHAIR: Just one question for Dr.
20 Rachman. Is it an emergency suspension or emergency
21 cancellation?

22 DR. RACHMAN: Emergency suspension, Madam
23 Chair.

24 MR. CASSIDY: Sorry, Madam Chair.

25 The next section deals with the EPA

1 status of pesticides approved for forestry use in
2 Ontario and we will be referring to a table which can
3 be found in the witness statement at page 21, which is
4 now on an overhead, and as Dr. Rachman indicated, she
5 would like to make a slight correction to the table
6 which she will indicate to you now.

7 DR. RACHMAN: Shall we do the correction
8 first?

9 MR. CASSIDY: Whatever is best for you.

10 DR. RACHMAN: Well, since we have already
11 discussed it I might as well refer to those...

12 MR. CASSIDY: This is Table 1, Madam
13 Chair.

14 DR. RACHMAN: The correction I wish to
15 make is in the right-hand column under fenitrothion. I
16 would like to insert the example that I gave you
17 earlier in my testimony about the interim re-entry
18 requirement imposed for greenhouse and nursery uses of
19 fenitrothion pending submission of exposure data.

20 MR. CASSIDY: I am indicatig right on the
21 right-hand corner for your benefit, Madam Chair, where
22 that change should be made of Table 1.

23 Q. Dr. Rachman, if you could just speak
24 up just a bit so that the people at the back of the
25 room can can hear you.

1 DR. RACHMAN: A. Certainly.

2 Q. Thank you.

3 A. Would you like me to repeat that?

4 Q. Yes, I think Ms. Seaborn needs that
5 again.

6 A. What I would like to have entered
7 there is the fact that an interim re-entry interval of
8 24 hours for greenhouse and nursery uses of
9 fenitrothion was imposed pending the receipt of
10 exposure data.

11 Q. That is, again, a non-forestry use;
12 is that correct?

13 A. That's correct.

14 MADAM CHAIR: What year was that, please?

15 DR. RACHMAN: I will have to check on
16 that for you, Madam Chair. Well, the registration
17 standard was 1987.

18 MR. CASTRILLI: Excuse me, Madam Chair,
19 may I ask the witness to repeat the very last portion
20 of that comment. It was pending receipt of...?
21 I didn't catch the remainder.

22 DR. RACHMAN: Exposure data. The
23 agency's concern was that available information was not
24 sufficient to determine whether greenhouse and nursery
25 workers were at risk, so they asked for exposure data

1 to be developed and until such time as they can
2 evaluate it, they imposed this interim re-entry
3 interval.

4 Now, let me emphasize that our review of
5 the available information showed that no human health
6 risks have been identified with respect to forestry
7 uses of the chemical pesticides approved for use in
8 Ontario and, therefore, no -- there have been no
9 cancellation proposals, no suspensions and no interim
10 risk reduction measures have been imposed.

11 I felt, though, that that would make a
12 pretty boring chart if it was all nos, so what I did
13 was to include, just for purposes of your information,
14 some of the risk reduction measures that have been
15 imposed for other uses so that you can see the kinds of
16 things that the EPA has done for some of these
17 chemicals.

18 Before we start, let's just clarify once
19 more the registration status of a couple of these
20 chemicals. First of all, aminocarb or Matacil is no
21 longer registered in the United States. There were no
22 regulatory actions against it, as far as we could
23 determine. My conclusion is that it was a voluntary
24 cancellation on the part of the registrant and I do not
25 know why. I would expect it had to do with some market

1 consideration, but I really can't say for sure.

2 Fenitrothion is not registered for food
3 use. It is registered for forestry uses, it is also
4 registered for use on -- I'm afraid I can't remember
5 the exact terminology, it is domestic and commercial
6 and industrial insecticidal use, so it's registered for
7 use around the home for insecticidal purposes, but not
8 on foods or in food handling areas.

9 All of the other chemicals are registered
10 for food uses. So that means that in putting together
11 the registration standards, EPA is evaluating these
12 chemicals with respect to the most stringent and
13 extensive data requirements of 40 CFR, Part 158. The
14 registration amendments column applies to uses other
15 than forestry uses since there have been no actions
16 against the forestry uses.

17 Now, we can take these in turn. 2,4-D.
18 When we prepared our statement of evidence, this
19 special reviews that had been proposed in 1986, EPA
20 decided in March of 1988 that the available evidence
21 just did not warrant the initiation of a special
22 review.

23 Since we prepared this statement of
24 evidence, we became aware of a subsequent notice
25 published in the Federal Register in October of 1989

1 and in that notice EPA reserves the right to initiate a
2 special review and announces that it will postpone
3 making the decision as to whether or not a special
4 review is warranted until it receives two epidemiology
5 studies which were currently underway at that time and
6 which it had become aware of.

7 MR. CASSIDY: Q. And I understand you
8 wish to file a copy of that notice?

9 DR. RACHMAN: A. Yes.

10 MR. CASSIDY: This would be exhibit, I
11 believe, 1242, Madam chair, entitled A Notice of Status
12 of Consideration for a Special Review. (handed)

13 MADAM CHAIR: Thank you.

14 ---EXHIBIT NO. 1242: Document entitled Notice:
15 Status of Consideration for a
Special Review.

16 DR. RACHMAN: Now, that notice which has
17 just been filed as an exhibit sets out some deadlines
18 by which the agency expected to receive these studies.
19 To the best of my knowledge, those studies are still
20 not available as of last week, in fact that was what
21 Environ was able to determine.

22 Now, may I ask see a copy of that notice?

23 MR. CASSIDY: Certainly.

24 DR. RACHMAN: Thank you. Let me just
25 make sure that my...

1 On the last page of the notice EPA says
2 that they are waiting for two NCI, National Cancer
3 Institute, case control studies. The first study from
4 eastern Nebraska has been completed but results will
5 not be released to EPA until the end of 1989. As far
6 as I know those results have still not been released.

7 Then the notice goes on to say:

8 "Statistical analysis of the second
9 study, from Iowa and Minnesota, is in
10 progress, with release anticipated in
11 March of 1990."

12 We were unable to determine whether that
13 study has been released yet. We think it has not.

14 So EPA is going to postpone its decision
15 as to whether or not to initiate a special review until
16 such time as it has an opportunity to review those
17 studies.

18 MADAM CHAIR: Excuse me, let me get this
19 straight. In the chart that we are looking at, the
20 special review proposed in 1986, a decision was made...

21 DR. RACHMAN: Not initiate in 1988.

22 MADAM CHAIR: And then in 1989 they
23 decided they would -- they might initiate a special
24 review depending on --

25 DR. RACHMAN: That's correct. What they

1 are saying essentially is that we've heard that there
2 is some new evidence on the way, this evidence may in
3 fact trigger a special review, we can't tell until we
4 see it, we will let you know when we see it, whether
5 the evidence supports the initiation of the special
6 review or not.

7 MR. CASSIDY: And that is the effect of
8 Exhibit 1242?

9 MADAM CHAIR: That's correct..

10 MADAM CHAIR: So what was added to Table
11 1 in the witness panel, Exhibit 1240?

12 DR. RACHMAN: That's correct.

13 MADAM CHAIR: Okay.

14 DR. RACHMAN: Now, we should point out,
15 there are also some animal studies relating to the
16 carcinogenicity of 2,4-D that are outstanding and yet
17 to be completed.

18 However, my interpretation of this notice
19 is that the EPA is not going to wait to receive the
20 data from these animal studies in order to make the
21 decision as to whether or not to initiate the special
22 review, that decision will be made based on these
23 epidemiology studies which should be available well in
24 advance of the animal data.

25 Now, just let me belabor this point a

1 little bit more. What we are talking about here is the
2 initiation of a special review of a risk benefit
3 determination, not a cancellation. And I hope I've
4 made it clear in my testimony the relationship between
5 the two, and the fact that what the agency is talking
6 about initiating for 2,4-D is an indepth review and
7 would probably take several years to conclude.

8 MR. CASSIDY: Q. Dr. Rachman, with that
9 last comment in mind, I would ask you to refer to the
10 third page of Exhibit 1242 in the bottom left-land side
11 of the conclusion. The photocopy seems to have made a
12 glitch there, but can you read the last sentence on
13 that section for us?

14 A. "In making the determination...."

15 Q. Yes.

16 A. It says:

17 "In making the determination to defer its
18 decision on initiation of a special
19 review, EPA has concluded continued
20 registration of 2,4-D, 2,4-DB and 2,4-DP
21 does not pose an unacceptable risk..."

22 The word is missing, but...

23 Q. I think it is "during".

24 A. "...during the time it will take to
25 receive the new studies and make a

1 comprehensive evaluation."

2 Q. All right. Thank you.

3 MADAM CHAIR: So they are not putting
4 into place any conditions with respect to the
5 registration?

6 DR. RACHMAN: No, that's correct. That's
7 correct.

8 MR. CASSIDY: I believe that concluded
9 yours evidence, Dr. Rachman --

10 DR. RACHMAN: In fact, it does not.

11 MR. CASSIDY: I'm sorry.

12 DR. RACHMAN: I would like to say a few
13 more things. I will be brief.

14 MR. CASSIDY: We are breaking at 10:10;
15 is that correct, Madam Chair?

16 DR. RACHMAN: I will be finished before
17 that, I promise.

18 MR. CASSIDY: I am not trying to put a
19 deadline on you, Dr. Rachman, I just give you the
20 parameters.

21 DR. RACHMAN: Really, I have almost ran
22 out of steam here.

23 I think the other entries on this table
24 are self-explanatory. The only other chemical
25 pesticide that has ever been even considered for

1 special review is carbaryl. That was way back in the
2 late 1970s and there again in 1980 the agency published
3 a notice that the available evidence just did not
4 support the initiation of special review.

5 I do not recall what the issue was at
6 that point. There was a toxicity issue, but I'm sorry,
7 I just don't remember what the details were.

8 MADAM CHAIR: So the initiation of this
9 special review of the risk/benefits of 2,4-D is
10 applicable only to the agricultural use of the product,
11 not to a forestry use of 2,4-D, or obviously if they
12 find some risk in the agricultural side--

13 DR. RACHMAN: That's right.

14 MADAM CHAIR: --it would extend to other
15 uses.

16 DR. RACHMAN: If the question is the
17 toxicity of the material, it is in the case of 2,4-D,
18 they will examine all the uses and all the exposures to
19 see what the risks are under every condition of
20 exposure that's allowed by the current registration, so
21 the forestry use will certainly be considered.

22 MR. MARTEL: They would allow use of a
23 product in forestry simply because of the infrequency
24 at which it would be used as opposed agriculture?

25 DR. RACHMAN: Well, that might turn out

1 to be the case, Mr. Martel.

2 MR. MARTEL: I am not saying they are
3 constant, just based on the frequency of the
4 application of it?

5 DR. RACHMAN: Right. Dr. Rodricks will
6 be talking about these sorts of issues in his
7 testimony. When you are looking at cancer risk, the
8 extent of exposure over a lifetime is a very important
9 variable.

10 So you are absolutely right, they would
11 make take a look at the extent of lifetime exposure and
12 make the determination of lifetime cancer risk, and it
13 might happen that the cancer risks for certain uses,
14 perhaps for forestry, would be within an acceptable
15 level, whereas the risks of certain other uses, perhaps
16 the agricultural uses, would not. That is the domain
17 of risk assessment and that's what Dr. Rodricks will be
18 talking about.

19 I would like to point out one other thing
20 which is not on this chart but just for your
21 information. The EPA maintains a categorization system
22 for chemicals that are thought to be carcinogenic and
23 its what's called a weight of evidence classification
24 and it's a convenient ranking scheme to express the
25 nature and the quality of the evidence of

1 carcinogenicity on those various chemicals.

2 Now, being on this list implies nothing
3 about risk, we are talking strictly about the inherent
4 toxicity of a chemical, strictly the evidence that it
5 causes cancer. Whether or not it causes a risk under
6 use conditions is, again, a subject for risk
7 assessment, it depends on the exposure under use
8 conditions of a chemical. So what we have here is
9 just -- it's a classification scheme.

10 Since we prepared our statement of
11 evidence, two of the chemicals of interest here have
12 changed classification and I'm not going to go into any
13 great detail on the scheme, it will probably be more
14 appropriate for Dr. Rodricks to do deal with that
15 later, but I will just explain the two changes that
16 have occurred.

17 MR. CASSIDY: Q. And I understand you
18 wish to file a document which outlines those two
19 classifications?

20 A. Yes, that's correct.

21 MR. CASSIDY: This will be the next
22 exhibit, Exhibit 1243, Madam Chair, and if it can be
23 titled EPA Updates List of Classified Carcinogenic
24 Pesticides. That title you will see at the very -- on
25 the front of Exhibit 1243.

1 ---EXHIBIT NO. 1243: Document entitled EPA Updates
2 List of Classified Carcinogenic
 Pesticides.

3 DR. RACHMAN: Now, this exhibit is not an
4 official agency publication, it is a reprint of an
5 article I found in Pesticide and Toxic Chemical News,
6 which is the trade publication I mentioned before.

7 Apparently someone in Congress requested
8 an update from EPA as to the carcinogen classification
9 of currently registered active ingredients and EPA sent
10 this list over to Congress and it was reported in the
11 press. EPA will eventually issue an official
12 memorandum. They occasionally issues these lists, not
13 on a regular basis.

14 MR. CASSIDY: Perhaps we can note for the
15 record that this update, Exhibit 12423, is dated May
16 2nd, 1990.

17 DR. RACHMAN: Now, the two items of
18 interest here are 2,4-D -- I'm sorry, there are three,
19 2,4-D, glyphosate and simazine.

20 2,4-D and glyphosate have moved to the D
21 category. The D category is the one that essentially
22 means we don't have enough information to tell whether
23 or not this is a carcinogen or not. We have required
24 new studies, we are waiting for the results.

25 Simazine is listed as category C.

1 Category C is the one that entails limited evidence of
2 carcinogenicity. This means that there were tests
3 performed in two species, rat and mouse. This simazine
4 was positive in only one, only in rat and is not
5 carcinogenic in the mouse, and I believe category C
6 also implies that the material is not a genotoxic.

7 Is that correct, Dr. Rodricks?

8 DR. RODRICKS: Yes.

9 DR. RACHMAN: Does not cause mutations in
10 mutagenicity studies. So the designation for chemicals
11 in category C is possible human carcinogen which
12 reflects uncertainty as to whether the animal evidence
13 indicates that it is a potential human carcinogen or
14 not. I just wanted to bring those to your attention.

15 MADAM CHAIR: Now, to clarify this, a
16 congressman has asked that this be updated?

17 DR. RACHMAN: Yes.

18 MADAM CHAIR: And has EPA done --
19 reclassified these?

20 DR. RACHMAN: Well, they have -- yes,
21 they have updated their list. They maintain a list of
22 some kind and there is an ongoing evaluation process.
23 As new data come in on various chemicals in the
24 registration standard process, these categories may be
25 changed to reflect the latest data and this list

1 doesn't appear with any regular frequency, it is not a
2 mandated list, but usually once or twice a year they
3 update to reflect new categories -- or changes in
4 categories that have been assigned.

5 I might also point out that the science
6 advisory panel gets involved in reviewing these
7 qualifications. So the EPA will propose a
8 classification for a chemical based on its review of
9 new studies and present that to the SAP and the SAP
10 will, you know, invite comment from the public and
11 evaluate the information and either concur or disagree
12 with the agency classification.

13 MR. CASSIDY: All right. Just one final
14 matter to be dealt with before the break, Madam Chair.

15 The evidence refers to -- the witness
16 statement refers to the farm mortality study which, you
17 may recall, was also discussed by Dr. Ritter in his
18 evidence back in MNR's Panels 12 and 13. You may
19 recall that at the time Dr. Ritter gave his evidence
20 and indeed at the time this witness statement was
21 written, the farm mortality study was only available in
22 an abstract form.

23 It is referred to on page 62 of the
24 witness statement, but since the publication of this
25 witness statement and the evidence of Dr. Ritter, that

1 study has now become available and just before we break
2 I would like to provide you and the parties with a copy
3 of it and it will be discussed by Dr. Rodricks briefly
4 in his evidence, and if that could be Exhibit 1244,
5 being the Saskatchewan Farm Mortality Study. I have
6 paraphrased the title because it would take the length
7 of the break to read the title, so I will just call it
8 that.

9 MADAM CHAIR: What's the date on that?

10 MR. CASSIDY: It is dated April 4th,
11 1990.

12 MADAM CHAIR: Who are the authors?

13 MR. CASSIDY: The authors are Wigle et
14 al. (handed)

15 MADAM CHAIR: Thank you.

16 ---EXHIBIT NO. 1244: Document entitled Mortality Study
17 of Canadian Male Farm Operators:
18 Non-Hodgkin's Lymphoma Mortality
and Agricultural Practices in
Saskatchewan.

19 MADAM CHAIR: Please remind me, for whom
20 was this study done? Was it for the National Cancer
21 Institute or the Federal Government in Ottawa?

22 MR. CASSIDY: It was --

23 DR. RACHMAN: I am afraid I don't
24 remember.

25 MR. CASSIDY: It was carried out -- Dr.

1 Ritter was one of the participants. It is a Canadian
2 study and I believe was carried out through the
3 Laboratory Centre for Disease Control, although I am
4 not certain of that, Madam Chair, and it may be helpful
5 to go back and review the evidence of Dr. Ritter.

6 MADAM CHAIR: Okay, thank you.

7 MR. CASSIDY: It is a Canada-wide study.
8 However, Saskatchewan was, as you will recall from Dr.
9 Ritter's evidence, and as is referred to in the
10 abstract, the first province in Canada for which data
11 was produced.

12 It might be appropriate now for the
13 break, Madam Chair, and we will commence after it with
14 Dr. Rodricks' evidence.

15 MADAM CHAIR: Perhaps you could have the
16 witnesses just review the study quickly over the break
17 and let us know for whom it was done. That will save
18 me going back to looking at the Ritter evidence.

19 DR. RACHMAN: Certainly.

20 MR. CASSIDY: All right.

21 MADAM CHAIR: Thank you, Mr. Cassidy.

22 The board will be back in 20 minutes.

23 ---Recess taken at 10:15 a.m.

24 ---On resuming at 10:45 a.m.

25 MADAM CHAIR: Please be seated.

1 Mr. Cassidy, just a short announcement.
2 We have a new telephone number and new fax number if
3 the parties want to take this down. Our telephone
4 number here at 151 Bloor Street is 963-1249. That's
5 963-1249, and the new fax number (416) 963-1252.

6 MR. CASSIDY: Does that fax come into
7 these premises, Madam Chair?

8 MADAM CHAIR: Yes.

9 MR. CASSIDY: Thank you. Madam Chair,
10 does the Board intend to announce today the schedule
11 for Panel 10, the OFIA panel?

12 MADAM CHAIR: Later today or tomorrow
13 morning.

14 MR. CASSIDY: Thank you.

15 MR. FREIDIN: Madam Chair, just on that
16 point. I can advise that if the Board wishes to
17 commence a week earlier than the 13th that poses no
18 problem for the Ministry.

19 MADAM CHAIR: Thank you, Mr. Freidin.

20 MR. CASSIDY: Madam Chair, we are now
21 prepared to commence with the second section of this
22 evidence and the second witness, Dr. Rodricks, who is
23 before you. For your notes, this section commences at
24 page 24 of the witness statement, Exhibit 1239, through
25 to the conclusion of the evidence, page 73.

1 Dr. Rodricks will be referring to a
2 number of overheads, which have been filed as Exhibit
3 1241, in the course of the presentation of his
4 evidence. I will turn on the first overhead which can
5 be found at page 1 and Dr. Rodricks will commence by
6 discussing the various approaches to health risk
7 evaluation which are outlined on that overhead and in
8 the evidence.

9 DR. RODRICKS: Thank you, Madam Chair.
10 We were asked to discuss general approaches now taken
11 for the evaluation of human health risks from
12 substances in the environment, and to define some of
13 the terms of that evaluation, at least as they are used
14 in the United States, and then to look more
15 specifically at some evaluations that have been
16 reported from various groups on pesticides used in
17 forest settings.

18 So I am going to proceed, then, through
19 each of those steps by beginning with a general
20 discussion of the process of risk assessment as it's
21 called, or risk evaluation as I call it here, and I
22 think it is important to do this. This may be quite
23 familiar to many of you, but I find that there is
24 sometimes confusion with respect to certain terms and
25 their use in the process.

1 I might also say that the terms that I am
2 going to describe here came out of a review conducted
3 by the National Academy of Sciences in the United
4 States, 1983, a review of what had been going on in the
5 federal government with respect to the evaluation of
6 risks from chemicals in the environment.

7 The academy committee that looked at the
8 issues said that there is a consistency in approach,
9 there has been for quite a long time, but not a good
10 consistency in the use of terms and they proposed a
11 specific way in which risk assessors in government and
12 outside of government ought to organize information and
13 to have it proceed through the evaluation process, and
14 the committee noted that whether one was talking about
15 a chemical risk or any other kind of risk, that there
16 is a general procedure, a way to think about the
17 problem and to organize your evidence.

18 So this procedure, which includes a
19 four-step process which I will go through here, is now,
20 I would say, used, at least in the United States, in
21 almost every situation where individual -- where either
22 governments or other individuals are looking at the
23 question of potential risk.

24 I might say that in other areas of the
25 world, I am familiar with what what goes on in this

1 kind of evaluation, there are some differences among
2 countries in the use of certain terms and there are
3 even some analytic differences in the way certain
4 problems are approached and I will point out one of
5 those, but generally you will find that in any
6 evaluation these same steps are followed. They may be
7 called other things and may not appear, as I said, in
8 this stepwise fashion, but it is the general approach
9 toxicologists follow.

10 The first step of the process, what we
11 call hazard identification, concerns the issue -- first
12 of all, by hazards we mean any inherent dangerous
13 property, in this case the chemical agent. Some agents
14 are highly flammable, some chemicals are radioactive,
15 we are particularly concerned with the issue of
16 toxicity. The first step in evaluation is to survey
17 scientific -- after you have identified the agent in
18 which you are interested, to survey the scientific
19 literature and attempt to identify --

20 MADAM CHAIR: Excuse me, Dr. Rodricks,
21 can we just have a short break.

22 MR. CASSIDY: They have to report a
23 spill.

24 MADAM CHAIR: I did the same thing
25 myself, Mr. Castrilli, this morning.

1 MR. CASTRILLI: Madam Chair, let the
2 record show that I was not the source of the spill, I
3 was simply the victim of the spill.

4 MR. CASSIDY: I think the source of the
5 spill was a representative of the Ministry of the
6 Environment.

7 ---Discussion off the record

8 MR. CASSIDY: Just while we are waiting,
9 it might be an opportunity for Dr. Rachman to advise
10 the Board with respect to the last question on the farm
11 mortality study. We have pulled the transcript of Dr.
12 Ritter's evidence and I think Dr. Rachman can interpret
13 it for the benefit of the Board.

14 DR. RACHMAN: Madam Chair, do I
15 understand your question correctly that you were
16 interested in the purpose for conducting the study?

17 MADAM CHAIR: No, who funded the study?
18 Was it --

19 DR. RACHMAN: Well, I'm not sure I can
20 answer that particular question, but I will read to you
21 from Dr. Ritter's testimony. This is Volume 122, page
22 20,463. The question to Dr. Ritter from Eleanor Cronk
23 was:

24 "Who was The author of this abstract?"

25 His answer is:

1 "There were five of us. These are the
2 joint collaborators on this study which
3 are drawn from the Centre for Disease
4 Control, the Laboratory Centre for
5 Disease Control of the Department of
6 Health and Welfare and the Environmental
7 Health Directorate, my group of the
8 Department of Health and Welfare."

9 And that's really all he says. I don't
10 see anything here specific to the funding of this
11 study.

12 MADAM CHAIR: That's fine. Thank you.

13 MR. CASSIDY: Q. I believe we are now in
14 a position to proceed Dr. Rodricks.

15 DR. RODRICKS: A. I was just beginning
16 to go through quickly the four steps of any complete
17 risk evaluation.

18 The first step, as I said, concerns
19 identification of the agent you are interested in, in
20 our case, for example, 2,4-D or other herbicides, and
21 to survey the scientific literature to identify the
22 kinds of toxicity, the specific agent seems capable of
23 causing under some conditions. All chemicals will
24 cause toxicity under some conditions, the important
25 point is what type of toxicity, does it cause cancer,

1 does it cause birth defects, does it effect
2 reproduction, does it irritate the skin, and those
3 properties of chemicals vary widely among them. So you
4 want to find out whether a chemical can cause - the
5 important word there is cause - specific kinds of
6 conditions under some conditions.

7 Now, the primary sources of information
8 for this, among toxicologists and risk assessors, come
9 from studies in experimental animals. The reason for
10 why resort to the use of experimental animals even
11 though that's not the rodent -- laboratory rodents are
12 not the species of interest to us ultimately, is that
13 these are studies that have two pretty, I should say,
14 characteristics that make them very, very important.

15 No. 1, you can gather information on
16 toxicity before you permit human exposure to take
17 place. That's very, very important. No. 2, if animal
18 studies are well done, it is possible, relative easy
19 and possible to establish causation in a fairly
20 straightforward way; that is, if you do the experiment
21 well, you have animals that receive the agent of
22 interest at various levels and then a set of control
23 animals that are identical in every single respect
24 except they do not receive the agent of interest, so if
25 there is an effect you can attribute causation fairly

1 easily.

2 Then the third important point is,
3 although it is not perfect, we believe animal results
4 are applicable to people with some exceptions and some
5 qualifications, but there is enough basis for believing
6 that if you see a carcinogenic response in animals that
7 is convincing, there is a reason to be concerned that
8 the agent may also present a cancer hazard to people.
9 Again, that's not a perfect -- that's not perfect
10 knowledge, but we behave as if that's the case
11 generally.

12 Epidemiology studies - and I will come
13 back to this point in a little more detail later - are
14 obviously of great importance because they are studies
15 in human beings. The problem is that, No. 1, these
16 studies are not controlled studies in any sense of the
17 word like a laboratory study and it is a very, very
18 difficult to establish a truly something approaching in
19 a truly controlled situation. I will come back to that
20 point a little bit later.

21 Q. The farm mortality study is an
22 epidemiology study; is that correct?

23 A. Yes, and there are, as I will show
24 later, 15 to 20 epidemiology studies on, if not 2,4-D
25 itself, at least the broad class of herbicides.

1 Anyway, it's very difficult -- the point
2 I was just going to make is that it's very difficult to
3 establish causation from epidemiology studies because
4 you don't have a truly controlled situation. You are
5 taking advantage of a situation where people are
6 already exposed and trying to set up something like a
7 controlled study, but it's not really achievable with
8 any single study.

9 I might say that for chemicals in
10 general, most of the information that is the basis for
11 regulation, certainly for pesticides and for almost all
12 other chemicals, comes from animal studies. There are
13 hundreds of epidemiology studies in the literature on
14 chemical agents mostly in occupational settings where
15 you can have more intense exposures and a lot of those
16 show some kind of association between exposure and some
17 health conditions or cancer even, but there are only
18 about 30 where 30 chemicals or substances, mixtures of
19 chemicals where the evidence has arisen to the level of
20 what we would call a causal relationship, and I will
21 come back to that point later.

22 At any rate, you need to look at both
23 kinds of studies when you are looking at a particular
24 chemical and make then a judgment about whether a
25 causal link exists. The second step, which I will deal

1 with more briefly now because I have a chart to
2 illustrate this one in a couple of overheads later, we
3 are trying to understand the relationship between
4 exposure, the magnitude of exposure to the chemical,
5 what we call the dose in sort of technical jargon, and
6 the frequency and severity of adverse health effects
7 because one of the principles of toxicology that not --
8 the dose, the size of the dose determines how much risk
9 there is for any agent, and I will come back to that
10 point later, but we are trying to understand that
11 relationship for each of the toxic effects of concern.

12 The third step, then, is what is called
13 the human exposure evaluation. The first two are
14 specific to the chemical and come out of the scientific
15 literature. The third step concerns the specific uses
16 of the chemical that you might be interested in. In
17 our case, the pesticides used in forest settings, we
18 would like to know that given the conditions of use of
19 those chemicals, who might come into contact with them,
20 how much of those chemicals get into peoples' bodies or
21 onto their bodies, how much of the chemical over what
22 period of time. We would like to know the answers to
23 all of those questions because all of these determines
24 how much exposure, how much risk might arise.

25 I hope you can see at this point that the

1 fourth step, what I have called -- the Academy of
2 Science has called the risk characterization step
3 involves integration of information from those first
4 three. There is no new information here in this step,
5 it's rather putting together all of the information in
6 the first three steps: What kind of health effects,
7 how do they relate to exposure, what is the actual
8 extent of exposure, and putting that together you
9 arrive at some picture of the risk, the likelihood that
10 under specific conditions those harmful effects are
11 going to appear. That is called the risk.

12 I put down likelihood here and I will
13 show you a minute that how likelihood is expressed in
14 kind of qualitative terms, kind of a judgment, and it
15 might be expressed in more quantitative terms as well,
16 and I go into this in a little more detail on my second
17 chart.

18 One final point here, that these four
19 steps comprise the risk assessment or risk evaluation
20 process. They don't in themselves lead to a decision,
21 but the use of a particular chemical -- there is a
22 separate step in the process which we refer to as the
23 risk management step that involves policy, the question
24 of -- a decision about whether the risks are low enough
25 to be considered insignificant or are they too high,

1 are they a significant public health burden and then
2 other factors may also enter into a decision. As Dr.
3 Rachman pointed out in connection with FIFRA in the
4 U.S., benefits of the use of the pesticide are
5 considered, et cetera, but those are a separate step
6 and the risk analyst really doesn't answer those
7 questions for us, although there are a lot of
8 precedents that we can point out to for decisions about
9 acceptable or significant or insignificant health risks
10 based on this analysis.

11 Let me then move on to the second chart.
12 The Board asked during the scoping session some
13 questions about weight of evid -- so-called weight of
14 evidence evaluations or judgments versus more
15 quantitative evaluations of risk and I want to address
16 that briefly and the process.

17 Q. Looking at page 2 of Exhibit 1241.

18 A. Page 2. I do not see these as
19 either/or kinds of situations, but rather I believe a
20 full evaluation of risk deals with both what are sort
21 of qualitative weight of evidence evaluations, as well
22 as more qualitative evaluations.

23 Step one of the process, the hazard
24 evaluation, is largely a weight of evidence evaluation.
25 It is not easy to quantify this; we are answering the

1 question: How likely is it that agent "x" causes birth
2 defects and if we have animal evidence on that, we need
3 then to judge the quality of all of the evidence
4 available, we may then also look at human evidence and
5 make an overall weight of evidence evaluation on the
6 hazard question. So it's clearly a very important part
7 of that step.

8 The Ministry of the Environment panel of
9 experts report on 2,4-D that I will come back to in a
10 moment, is largely a weight of evidence evaluation on
11 the question: Is 2,4-D a likely human carcinogen under
12 any conditions. It is only the first step of the
13 hazard -- of the risk assessment process, it is only an
14 attempt to answer that first step, although the panel
15 also got into some risk issues as well, and I will come
16 back to that, but most of the report is this weighted
17 evidence kind of evaluation.

18 MR. CASSIDY: That MOE report is Exhibit
19 714, Madam Chair.

20 DR. RODRICKS: Steps 2 and 3, the
21 so-called dose response relationship and human exposure
22 evaluation, we try to make those as quantitative as we
23 can. There are some sort of qualitative judgments in
24 that process as well, but it is very hard to reach firm
25 conclusion without some quantitative information; how

1 much exposure do people have, that's more than a
2 qualitative question. You try to get some quantitative
3 information about the size of that. So that figure is
4 quite important.

5 Step 4, the final risk characterization.
6 I might say that the National Academy of Science panel
7 chose the term risk characterization very carefully
8 because they were concerned that given our state of
9 knowledge these judgments couldn't be strictly
10 quantitative in nature if we don't know enough, and
11 that there ought to be accompanying any quantitative
12 evaluation a kind of weight of evidence evaluation as
13 well. A qualitative discussion of how good the
14 evidence is and the uncertainties is not quantitative.
15 So I see these as not either/or kinds of evaluations,
16 but as really one in the same.

17 Now, I would also have to point out, and
18 this comes up a great deal in the Crump evaluation as I
19 will refer to it and that's Exhibit...

20 Q. 716.

21 A. There is uncertainty in any risk
22 evaluation. I have never seen one in all my years of
23 doing this that didn't have some uncertainties in it.
24 There are two kinds. You may not always have all the
25 data you want on a specific chemical or its uses, and

1 then there are also -- there are just some gaps in our
2 basic scientific knowledge on many of these questions,
3 that's another uncertainty. I will show you in the
4 next graph a very, very important example of that.

5 The general approach in the risk
6 evaluation process is where you have a range of
7 possibilities, scientific possibilities to choose from
8 and you really can't pin down at a particular step in
9 the analysis one or the other as better supported
10 scientifically. The typical procedure in the risk
11 assessment process is to choose that assumption which
12 will yield the highest estimate of risk, that is called
13 a conservative or worst-case kind of analysis.

14 Now, you don't have to do that
15 exclusively, we also like to give what is called a more
16 typical kind of assumption or what we call a more mid
17 range kind of assumption so that the decision-maker can
18 look at what is perhaps a more reasonable kind of
19 approach, but then you also want to look at what is the
20 worst, the most pessimistic view of what might happen.
21 The Crump analysis consistently uses, in at least one
22 of their analysis, a series of worst-case assumptions
23 that I will illustrate to go throughout the process
24 where you have certain kinds of gaps in knowledge.

25 The result of that is that - and I will

1 show a specific example in the next graph that I said -
2 we can't claim that the risks we produce are
3 quantitative estimates in particular, are accurate
4 portrayals of human risk. We don't have any way to
5 know that, at least from most exposures that occur in
6 the environment, but we can say - and this is EPA
7 language in the U.S. and I think quite widely
8 accepted - that by using this procedure we put what is
9 called an upper limit on the risk, an upper limit on
10 how bad the risk might be. It could be accurate but we
11 have no way to check it.

12 The actual risk is most likely less than
13 we're predicting by these methods and it could be zero,
14 but we don't know that either. Again, we can't have
15 perfect confidence, but high confidence that the risks
16 are like to be upper bounds.

17 If you look at the Board's review, they
18 made a point that you can't quantify human risk and I
19 would agree with that. We don't have the knowledge to
20 do that, but we can quantify some upper limit on what
21 it might be and we can be much more confident of that.

22 Let me give you one example of that on
23 the next chart which deals with this very tricky issue
24 of dose response evaluations.

25 Q. At page 2 --

1 A. A little sense of what goes on in the
2 process. This is a graph which plots risk, which I
3 have over near on the vertical axis, and the units
4 here -- let me just point to the units. With risks we
5 are dealing with probabilities. Remember I said in the
6 first chart it's the likelihood of some adverse effect,
7 that is the probability here. Probabilities range from
8 zero to one, they don't have any units. So we talked
9 about a one in ten chance of something happening or a
10 one in two chance or a one in a million chance or
11 whatever.

12 So I plotted over here on this axis the
13 figure 0.1, that's a one in ten chance of something
14 adverse happening, the risk is an adverse event, up to
15 something like 0.9, then I drew a line across here at
16 .1. I did that because -- and then I called this, the
17 region from risks of .1 and higher, the range of
18 observation and I will explain that in a second.

19 The other axis, the horizontal axis, is
20 the dose of the chemical, the exposure to the chemical
21 or dose, more technically, the amount that gets into
22 the body. If you are talking about -- this could apply
23 either to animal studies or to situations where people
24 are exposed to the chemicals.

25 All our techniques for discovering

1 adverse effects are limited to quite high risk
2 situations. It is just, as a statistical matter, very
3 difficult or impossible to detect risks below about one
4 ten, that's a pretty high risk, but with animal
5 studies, unless you are going to use million and
6 millions of test animals or with human studies, human
7 studied tend to be even less sensitive, you have a
8 limit of the risk you could detect. You just run out
9 of -- it's like an analytical chemistry method, you
10 just run out of statistical power to detect a risk.

11 So I have called this the range of
12 observation and one of the reasons in the animal tests
13 you use very high doses, you use very high doses, much,
14 much higher than people would ever be exposed to, to
15 see if a risk can be produced and you do that because
16 you are limited to detecting very, very high risks.

17 So you will have -- I drew a straight
18 line here as the sort of dose response relationship and
19 I called that the observed response. I don't mean to
20 imply that every observed response is a straight line,
21 most of them are not, so this is kind of hypothetical,
22 but that's what you typically observe mostly from
23 animal studies.

24 I would also add that in human studies,
25 and it is a problem with every single one of the

1 studies we have on phenoxy herbicides in human studies,
2 we really do not have any quantitative dose information
3 from those studies at all. As a matter of fact, one of
4 the serious problems is that we are not even sure which
5 chemicals are involved in most of those studies. I
6 will come back to that.

7 So I am not talking here about a specific
8 chemical, but the general process because later in my
9 discussion I shall be referring to some of the terms
10 that come out in this discussion.

11 Now, if we look at most chemicals that
12 enter the environment and the pesticides at issue here
13 would be good examples, actual human exposures will
14 tend to be rather low relative to those where you can
15 observe effects. They will be way down in this range
16 and that's quite typical. You may indentify the few
17 occupational situations where exposures may be high,
18 but most typically environmental exposures are very,
19 very much lower.

20 So the question is: If I can detect
21 risks at very high doses and very high risks, then is
22 there a risk and what is its size at very low doses,
23 and that gets you into what is called extrapolation.

24 The general thinking now among
25 toxicologists is that -- first of all, this so-called

1 dose response relationship isn't just going to end
2 right here, it is going to continue below that even
3 though you can't detect it. Then the question is: In
4 what shape does it take in this range of extrapolation.

5 There is strong evidence that for most
6 toxic effects this will fall off at some point to what
7 is called a threshold, a no-effect level, a threshold
8 dose for most toxic effects, this is quite widely
9 accepted, such that the risk really drops off to zero,
10 at some dose well above zero and you can determine that
11 dose experimentally.

12 In risk assessment now, this assumption
13 of a so-called threshold is now used everywhere that I
14 know of for all kinds of toxic effects except cancer,
15 and the general procedure here, the risk assessment
16 procedure, is to identify that no-effect level
17 experimentally and then ensure that there is some
18 margin of safety as the term is called, margin of
19 safety; people are protected from that range where you
20 get effects by a margin of safety and there are some
21 precedents for margins of safety for various kinds of
22 toxic effects, and I will come to those.

23 Cancer is more controversial. From the
24 1940s and later, there is some experimental work for
25 some substances capable of causing cancer; namely, the

1 radiation of various types, that this curve -- this
2 relationship between dose and response came down in
3 what is called a straight line or a linear relationship
4 and the risk didn't really disappear until you got to
5 absolutely no exposure.

6 In other words, there was some risk all
7 the way down under this straight line here, but did
8 fall off. The probability of cancer would decline. So
9 it is really -- as I said, the experimental evidence of
10 for that is very limited and mostly to radiation,
11 Certain kinds of radiation.

12 There is some evidence for certain kinds
13 of radiation that there might not be a threshold; that
14 is, the risk goes to zero only when the dose -- at an
15 absolute zero risk only when the goes to zero, but
16 falls off in a curve line like this so that the curve
17 line -- if that were accurate at a given dose, you see
18 the curve line shows less risk than the straight line.
19 That's true all the way up.

20 Then there is also evidence for some
21 agents that even carcinogens may follow a threshold
22 kind of phenomenon. Again, this is an area of
23 uncertainty. The general approach, unless you have
24 very compelling evidence otherwise, is to assume that
25 all carcinogens, whether they are animal carcinogens or

1 known to be human carcinogens, follow this straight
2 line relationship.

3 I know of no evidence, I know of no
4 analysis which suggests the risk could be higher than
5 that straight line; that seems very, very improbable.
6 There is substantial evidence that it could be less and
7 it could be zero, but the general approach in risk
8 assessment, unless you have some data on sort of a
9 chemical specific basis, is to use the linear, no
10 threshold model, so it's the most pesimistic of what
11 could happen and this indeed what Crump did in his
12 analysis and this is indeed what the Ministry of
13 Environment expert panel did in their analysis.

14 I don't disagree with that, we just have
15 to be careful interpreting the results.

16 Notice that -- one final point here or
17 actually two small points. No. 1, when you follow the
18 straight line analysis for a carcinogen -- I said for
19 non-carcinogens what the risk assessment usually
20 reports is the margin of safety; that is, the
21 difference between the human exposure and the minimum
22 toxic dose or the agent.

23 For carcinogens, what is reported when
24 you adopt this quantitative procedure, this straight
25 line, is to report -- let's say, you determine the

1 lifetime dose - this is really the lifetime dose we are
2 talking about here - to be at some point along here,
3 you then use the procedure to go up to the line and
4 report the risk corresponding to that dose. So you
5 will see in Dr. Crump's report and the MOE report
6 presentations of risk as probabilities, mostly very,
7 very low probabilities, done by that basic or that sort
8 of analysis. So the risk is this probability of one in
9 ten million, one in a million, et cetera, et cetera, et
10 cetera.

11 As I said, there are precedents to turn
12 to to decide at what point you are going to say this
13 risk is really negligible, there is no real need as a
14 public health matter to worry about it. There are a
15 lot of precedents, at least in the United States, for
16 that, but that really is a separate risk management
17 decision.

18 The final point on this is that this
19 linear, no threshold model and this quantitative
20 expression of risk is used almost everywhere in
21 regulation in the United States for carcinogens as a
22 guide to regulation. As far as I know, it has not been
23 widely used at least in other countries, in Europe, in
24 Japan, in Canada. I know it has been discussed and
25 there have been some examples of its use. I'm not sure

1 why that is because in many cases more weight of
2 evidence kinds of judgments -- this threshold kind of
3 approach might be seen as kind of a weight of evidence
4 judgment and is used for carcinogens. Both of them
5 have merits and both of them have weaknesses, but in
6 the United States this procedure -- I feel fairly
7 confident it's a meaningful procedure as long as you
8 are careful to note what you are doing and the
9 uncertainties in it, that you are not really predicting
10 actual risk, there is really no way to know that.

11 Let me move on to the two specific issues
12 that we are going to go over in a little more detail
13 now, that is the general background and the context for
14 dealing with them.

15 Q. That is Exhibit 3 of Exhibit 1241.

16 A. As I said before, we were asked to
17 examine primarily an expert panel report of the
18 Ministry of Health, dated March 23rd, 1987 which
19 undertook a review on the question of the
20 carcinogenicity of 2,4-D specifically.

21 Then we were also asked to look at a very
22 extensive document that was prepared by Dr. Kenny Crump
23 and his associates. Dr. Crump has a small firm in the
24 United States, very much like our own, and they do
25 similar kinds of analysis. We had never seen this

1 report before when we were asked to review its content
2 to see if it conforms to good practices and risk
3 assessment. So I have some opinions on each of those.

4 I might just emphasize, as I tried to
5 before, that in the weight of evidence concerning the
6 carcinogenicity of 2,4-D we are talking primarily about
7 the question of whether it is -- whether it is known to
8 cause cancer under any conditions, the hazard step, not
9 the question of: Does it pose a risk, let's say, in
10 forest settings. That's a separate question that
11 requires -- if you decide in the first case that it
12 does cause cancer, then you would proceed with the
13 other steps to see how big the risk might be.

14 No. 4. I guess I need to add here, I
15 guess it would be the appropriate point, that since our
16 statement of evidence was prepared a second review of
17 the weight of evidence to carcinogenicity of 2,4-D has
18 been prepared by a group at Harvard University.

19 MR. CASSIDY: I have a copy of that
20 document to file. It is titled the Weight of the
21 Evidence of the Human Carcinogenicity of 2,4-D. It
22 will be Exhibit 1245.

23 ---EXHIBIT NO. 1245: .Document entitled The Weight of
24 the Evidence of the Human
25 Carcinogenicity of 2,4-D.

1 DR. RODRICKS: This report appeared in
2 January 1990 and it was put together by Dr. John Graham
3 at the Harvard School of Public Health. He convened a
4 panel of experts in epidemiology and toxicology to look
5 at the question of the carcinogenicity of 2,4-D, the
6 hazard evaluation. So I have added this and some
7 conclusions from that report here.

8 Both of these groups looked at three
9 kinds of evidence and these are typically the kinds of
10 things you would look at in making this evaluation, the
11 available human epidemiology data, the available animal
12 evidence, then I noted here on this chart other
13 relevant data and by that I note -- and if you go
14 through these reports you will see that the experts
15 considered other information besides animal tests and
16 human tests as part of the evaluation. They looked to
17 see whether the material had any properties, for
18 example, to indicate it could damage genes or cells.

19 That is one characteristic, it is not
20 a -- whether it does nor not is not an overwhelming
21 determinant of whether it is a carcinogen, but it is an
22 important part of the evidence. They look at chemical
23 structure, other toxicology data besides the cancer
24 studies are relevant. So there was that kind of
25 analysis in each of these reports. Each of the two

1 evaluations looked at those three kinds of evidence in
2 great detail.

3 On the question -- on the human
4 epidemiology questions, I am going to repeat a couple
5 of points I made and go into them in a little more
6 detail on the general criteria for evaluating
7 epidemiology studies because, as I said before -- we
8 will go to overhead No. 5.

9 As I said before, these studies are not
10 controlled studies in the way a laboratory animal study
11 is controlled. So judging whether a causal
12 relationship - that's the ultimate question, do the
13 studies show a causal relationship, that's very, very
14 important - requires more than the usual kind of
15 analysis, and this is a source of great confusion.

16 There are, depending on how you count
17 them, 15 to 20 epidemiology studies reported since
18 19 -- most of them appearing in the last decade. On
19 phenoxy herbicides or -- that is the whole class of
20 about a dozen different phenoxy herbicides that have
21 been in use of which 2,4-D is one, and even herbicides
22 more generally, pesticides more generally or other
23 situations where exposures with these herbicides might
24 be involved. 15 to 20 such studies. That is the body
25 of evidence that these reports deal with.

1 I said already that these are not
2 controlled studies, but to varying degrees
3 epidemiologists are able to, by taking proper
4 precautions, set up something like a controlled
5 investigation where you are comparing individuals with
6 a disease to individuals without a disease and then
7 comparing the differences in their exposures or,
8 conversely, you might look at groups that have the same
9 kinds of exposures and follow them to see whether --
10 I'm sorry, you look at groups that have different
11 exposures, some to herbicides and some not to
12 herbicides, to see whether there is any different in
13 the disease pattern in those groups.

14 Those are the sorts of studies that are
15 undertaken and you try to set up something like a
16 controlled situation, but it is very, very rarely
17 achieved and achieved a true control situation in none
18 of these studies.

19 Now, another thing that is very important
20 to consider, and I am citing here some basic principles
21 that are I think widely accepted by epidemiologists
22 and, as far as I can tell, were used to guide the
23 evaluation of the Ministry of Environment group and the
24 Harvard group as well.

25 First of all, the appearance of what is

1 called a statistical association between exposure and a
2 health effect in a single study is not sufficient to
3 confirm a cause/effect relationship. There are
4 hundreds of epidemiology studies on pesticides and
5 many other chemicals where you see associations; that
6 is, in a given study there seem to be some excess of
7 the rate of a certain disease, certain cancers, certain
8 kinds of cancers and exposure to a particular chemical
9 or chemicals. For many, many reasons, just that single
10 observation would not be enough to establish causation.

11 I can use a simple example of why that
12 might be the cause. There is, for example, a strong
13 association between the number of hours people spend
14 watching television and the risk of coronary disease,
15 but we didn't really believe -- that's a very strong
16 statistical association, that televisions might cause
17 coronary heart disease, but it is more likely that
18 hours spend catching television reflects some other
19 factor, maybe reduced physical activity that may be
20 more directly related to it.

21 So that sort of association gives you
22 some clues and you can't ignore it, but, at the same
23 time, you can't jump to a conclusion of causation.
24 That's what we are trying to get to here.

25 And you see that, you see in the studies

1 on phenoxy herbicides, several examples in the
2 literature of associations between phenoxy herbicide
3 exposure and certain kinds of cancers. I could list
4 them. The earliest reports identified three kinds of
5 cancers as possibly associated with exposure to
6 phenoxyherbicides - these were reports from Sweden - of
7 a group of cancers called soft tissue sarcomas, STS,
8 and then two kinds of cancers of lymph cells.

9 I was going to write them down here but I
10 guess...

11 Soft tissue sarcomas, these are just
12 cancers of connective tissue of one type or another.
13 ,sarcoma is just one type of malignant cancer, and then
14 what is called Hodgkin's disease, which is a cancer of
15 certain lymph cells. It is a lymphoma, if you like,
16 and then non-Hodgkin's lymphoma, and pathologists make
17 a distinction between certain kinds of cells involved.
18 in Hodgkin's disease as cancers of the lymph glands,
19 lymph nodes and cells, and non-Hodgkin's lymphoma which
20 is basically all other kinds of -- I'm not enough of a
21 pathologist to distinguish, you need a pathologist to
22 distinguish them, but they do distinguish these as
23 different kinds of cancer that shouldn't be grouped
24 together.

25 Anyway, there were association reports

1 between exposures in Sweden among forestry workers,
2 people involved in spraying right-of-ways with phenoxy
3 herbicides back in -- these studies covered the period
4 79, '80 and '81 and these three kinds of cancer
5 associations were reported. That, of course, gave rise
6 to a series of studies over the next ten years and they
7 are still going on on these questions.

8 Now, because of the problem in any
9 particular study of distinguishing an association that
10 the statistician might see, any true causal
11 relationships, the epidemiologists seek - I have these
12 five criteria listed under Item No. 4 here - generally
13 seek before reaching a judgment about causation the
14 following kinds of evidence.

15 First of all, they look for consistency,
16 a consistent pattern of associations in several
17 studies, several undefined. There is no specific
18 criteria here, but you would like to see it in several
19 studies conducted in different populations by different
20 methods of study, and I will go back and review where
21 we are on phenoxies with respect to this criteria.

22 B, if you want to establish a causal
23 relationship, you ought to know what it is that those
24 people were exposed to in as much detail as you can;
25 what the chemical was, and how how much exposure

1 occurred and over what period of time. That's 4B.

2 C -- A and B are probably the strongest
3 components of the analysis. C is nice to have,
4 sometimes very difficult to apply and our
5 epidemiologists realize this, that just as in my dose
6 response relationship I showed you later -- I'm sorry,
7 earlier, you would like to see evidence that as
8 exposure increased in those populations the risk, the
9 numbers of cases, if you like, of the cancer also
10 increased.

11 There are many examples of instances
12 where you see an association between exposure and an
13 excess of cancer in one group, but then you look at a
14 group that is more intensely exposed for a longer
15 period of time and there is no association there. That
16 would raise great doubt. You wouldn't say there isn't
17 a causal relationship, but that sort of observation
18 would raise great doubt about whether there is a causal
19 relationship.

20 D, just the strength of the statistical
21 association is important here. How strong the
22 statisticians say this association is and then,
23 finally, some evidence from animal studies. This would
24 not be required, but if in fact you had confirmatory
25 evidence from animals, that would add further weight to

1 the evidence.

2 Now, these are general criteria and the
3 general procedure for reaching conclusions on any
4 specific case is to gather a group of epidemiologists
5 who look at all the available evidence and make a
6 judgment. This is not a simple formula that they might
7 carry through, they make a judgment about the weight of
8 that evidence and how closely or not so closely they
9 match these criteria.

10 In the case of 2,4-D in particular, I
11 will go to 5(a) which lists the conclusions and I will
12 tell you a few of the reasons why both the MOE experts,
13 Exhibit 714, and the Harvard panel of experts reached
14 the conclusions they did.

15 Q. That's Exhibit 1245, the Harvard
16 panel of experts.

17 A. This is only on the human
18 epidemiology evidence, it's not the total evidence. I
19 will talk a little bit again about animal evidence and
20 other kinds of effects.

21 With respect to the human evidence, the
22 MOE concludes, and I will read this quote and comment a
23 little bit about it and how it got there.

24 "Using IARC terminology..." Let me
25 clarify that. IARC stands for the International Agency

1 for Research on Cancer. It is a unit of the world
2 health organization in France and one of their -- they
3 do cancer research, but one other thing they do that's
4 very, very important is convene panels of experts to
5 review cancer data on environmental agents and other
6 agents as well, drugs and so forth, and reach
7 conclusions about the evidence of carcinogenicity.

8 They have three categories of evidence.
9 The first evidence is, we believe there is sufficient
10 evidence to establish causation and they have reached
11 that in about 30 cases. Some chemicals you may know,
12 benzene, arsenic, cigarette smoking, DES. As I said,
13 about 30 kinds of chemicals or, in some cases,
14 occupational settings.

15 They then have limited evidence, a
16 limited evidence category which says it does not rise
17 to causation but there is some suggestions of an effect
18 and further studies ought to be done, and then they
19 have inadequate to classify, that's the third IARC
20 category which says the evidence doesn't even suggest
21 even the limited category.

22 Okay. So the quote from the MOE is,
23 "Using IRAC terminology..." they chose
24 to use that same terminology,

25 "...it may be concluded that there is

1 limited evidence of carcinogenicity in
2 man from exposure to phenoxy herbicides.
3 In terms of exposure to 2,4-D
4 specifically, the evidence must be
5 regarded as inadequate to classify it as
6 a carcinogen."

7 If you read the report carefully, the
8 limited evidence they refer to primarily concerns
9 non-Hodgkin's lymphoma, although only one of these
10 three. The soft tissue sarcomas have been looked for
11 in several additional studies and seen one other time,
12 but in most cases you never see them again. So you
13 don't have any consistency.

14 Hodgkin's disease has not been seen with
15 any consistency whatsoever. There is some pattern of
16 consistency for non-Hodgkin's lymphoma, this particular
17 case, but it seems the best you can say is phenoxy
18 herbicides might be involved there, not any specific
19 herbicide, and it's suggestive only.

20 The Harvard panel of experts reaches
21 close to that same conclusion. There is one subtle
22 difference and I need to point that out. The Harvard
23 panel of experts' conclusion was, using case control
24 and cohort studies, those are two kinds of epidemiology
25 studies:

1 "Epidemiologists have examined whether
2 human exposure to phenoxy herbicides is
3 associated with various forms of cancer.
4 While a cause/effect relationship is far
5 from being established, the epidemiology
6 evidence for association between use of
7 2,4-D and non-Hodgkin's lymphoma is
8 suggestive and requires further
9 investigation."

10 Just my own editorial comment on that,
11 this report emphasizes throughout the problem in all of
12 these studies of separating 2,4-D from all the other
13 phenoxy herbicides. As you read the report, you find
14 them trying to deal with that problem. I just found it
15 a little surprising in the conclusion that they then
16 linked a suggestive evidence 2,4-D with non-Hodgkin's
17 lymphoma.

18 The rest of report wouldn't seem to
19 suggest that, but that's in fact what they say in their
20 conclusions. They suggest there is very little
21 evidence of association between use of 2,4-D and soft
22 tissue sarcoma or Hodgkin's disease and no evidence of
23 association between 2,4-D and any other form of cancer.
24 So those two conclusions -- there is a slight
25 difference between the two, but they are basically

1 very, very close. So causation not established, based
2 on human evidence, but certainly they are both
3 suggesting additional studies.

4 Animal evidence I will summarize quickly.
5 They all looked at that. The Crump report also deals
6 with the animal evidence. There are -- there were
7 three sets of studies. This is summarized in the Crump
8 report, published in the period from '69 up to through
9 '74 on 2,4-D and certain esters of 2,4-D and amine.
10 They were all negative, either in rats mice or both.

11 I must say that those three studies were
12 quickly passed by by the MOE Board of experts. They
13 are fairly old studies and although they don't show an
14 effect, you can't give them very much weight and I
15 wouldn't either.

16 There is a more recent study conducted at
17 EPA's requests by something called the Industry Task
18 Force on 2,4-D, that is a study in mice and rats of
19 carcinogenic activity in a lifetime feeding study for
20 2,4-D submitted to EPA. This has been reviewed and
21 judged as providing insufficient evidence of
22 carcinogenicity in mice and rats by the MOE panel of
23 experts and by the Harvard panel as well.

24 They did a very through evaluation in
25 both cases. The MOE panel's evidence evaluation is

1 essentially good on that particular question. EPA had
2 reached basically the same conclusions. There is one
3 thing you ought to know, and I think Dr. Rachman noted
4 it for you, the EPA is seeking additional animal
5 studies on 2,4-D and it is one of the controversies
6 that comes out of a technical issue having to do with
7 these cancer tests.

8 I said earlier that when you do a cancer
9 study you try to give very, very high doses so you can
10 be sure to predict risks if they are there because it
11 is difficult to predict risk unless you give very high
12 doses.

13 The EPA judged that the **ITF studies, at
14 least in the mouse and probably in the rat, do not get
15 to a high enough dose to be fully adequate to satisfy
16 their requirement, what is called a maximum tolerated
17 dose. The idea is you have got to pump up the dose
18 until you get as high as you can so you can see the --
19 but without threatening the lives of the animal in any
20 other way except for the development of the cancer. So
21 you try and increase the sensitivity.

22 EPA does not find them acceptable, the
23 MOE panel of experts found the rat study acceptable and
24 it is a very, very close call.

25 I have not gone through the studies in

1 all detail myself, so I do not have an independent
2 opinion on the -- the MOE panel thought it was achieved
3 in the rat but they were not sure it would have been
4 achieved in the mouse. They may not agree with EPA
5 when they evaluated the study, but that's all the
6 evidence we have and that's the conclusions reached by
7 those panels.

8 Finally I will go to a little bit of the
9 other relevant data. This was reviewed extensively by
10 the MOE panel of experts and less by the Harvard panel.
11 They didn't give it as much attention as the MOE panel
12 did. They have a long discussion of the so-called
13 potential for 2,4-D to cause damage to genes. These
14 are studies -- these are not cancer studies, but other
15 kinds of studies, some of them in test tubes with
16 bacteria, others in whole animals, and their
17 conclusion, the MOE conclusion -- there are probably, I
18 didn't count them, but there are probably two dozen
19 studies on 2,4-D in the scientific literature.

20 They decided it was not genotoxic, so
21 then finally putting all that together, both panels
22 agree on the total weight of the evidence from the
23 animals studies, the human studies and the general
24 toxicity studies. The MOE panel of experts said:

25 "There is insufficient evidence to

1 support a finding that 2,4-D is a
2 carcinogen and, consequently, there is
3 insufficient evidence to conclude that
4 existing uses of 2,4-D in Ontario pose a
5 significant human health risk."

6 I will mention in a moment, as I discuss
7 Crump now as my final discussion, that the MOE panel of
8 experts did go a bit beyond the weight of the evidence
9 evaluation of carcinogenicity and did some more
10 extensive evaluations of potential risk, and I will
11 refer to those as I proceed.

12 The Harvard panel of experts, I gave you
13 their conclusion on cause/effect relationships, it's
14 far being from being established. And, again, they do
15 suggest additional studies because of the possible
16 suggestion of an association with non-Hodgkin's
17 lymphoma that seems to appear in several studies; not
18 all studies, but several studies.

19 Q. The cause and effect relationship
20 they are referring to is between 2,4-D and cancer?

21 A. Yes, this is the whole -- that's what
22 these both refer to, a question of whether the hazard
23 evaluation -- whether 2,4-D is a potential human
24 carcinogen.

25 Q. Now, we are referring to page 9.

1 A. The last part of the presentation
2 deals with Dr. Crump's report, Dr. Crump and his
3 associates, Exhibit...

4 Q. 716.

5 A. 716. It is entitled Worst-Case
6 Analysis Study on Forest Plantation Herbicide Use. It
7 was prepared for the Department of Natural Resources of
8 the State of Washington. They're specific to
9 herbicides and at least three -- I have the seven that
10 they examined in detail and three, maybe four are
11 relevant here in Ontario, 2,4-D, glyphosate, and
12 picloram I understand are relevant here. I don't know
13 whether triclopyr is, I heard it might be, I don't
14 know. The others I guess are not, but these are the
15 ones that were of issue in the State of Washington.
16 I must say that they looked only at aerial application,
17 and I might emphasize that.

18 We went through the Crump analysis, it is
19 quite extensive, to determine whether it was conducted
20 according to standards that we believe are appropriate
21 for risk evaluation, the steps or the process I
22 referred to earlier, whether they were clear in the
23 assumption where they had to use assumptions about --
24 in certain cases, whether they stated what they were
25 and their basis and whether -- when they said they were

1 doing a worst-case analysis, whether they documented
2 that in fact that was the case. So that was our
3 analysis.

4 They evaluated a hazard identification
5 phase, all of the non-toxic effects of the herbicides.
6 There are extensive toxicity reviews throughout their
7 report. They did not discount any reported effect as
8 irrelevant to humans that we could see, and I think
9 there -- if anything, excessively concerned here.

10 There are some instances where the authors of some of
11 the reports that they reviewed concluded that a
12 particular effect was not related to the herbicide, but
13 it appeared and the authors may have tried to explain
14 it away as spurious, but in the Crump report you will
15 find consistently that they don't discount those
16 information. So they took a very conservative view to
17 the health evaluation.

18 They also did something that may sound a
19 little unusual, but it is not uncommon in risk
20 assessment, this third bullet, they assumed, even
21 without evidence, that all the herbicides could be
22 potential human carcinogens, even when the evidence was
23 negative or otherwise inconclusive. Now, why would you
24 do that.

25 Well, when you have a so-called negative

1 animal study, let's say, as you saw from my earlier
2 chart, I hope, that those studies are capable of
3 detecting fairly high risks at fairly high doses, but
4 it is just possible that there may be a cancer risk and
5 you couldn't detect it. So it is not uncommon in risk
6 assessment to say: Well, what if we just missed
7 detecting the cancer risk here, how bad -- a cancer
8 hazard, I should say, let me be careful, what if we
9 just missed it in this negative study, that there
10 really was an effect, but it was just below our
11 detection level.

12 Well, we can answer the 'what if'
13 question by doing a kind of risk assessment which says:
14 I am going to assume there was an effect right at the
15 detection limit of this experiment, and then proceed.
16 I will show you a little bit on the dose response how
17 that's done.

18 I might add a couple of points here. The
19 Ministry of the Environment panel had this -- I'm
20 sorry, the ITF animal study that I listed earlier, as
21 done by this Industry Task and submitted to EPA, was
22 not available to Crump when they did their analysis.

23 It was available to the MOE panel and
24 they reviewed it and did with it the same sort of thing
25 that Crump had done with the earlier cancer data; that

1 is, they assumed in their evaluation, they being the
2 MOE panel, that there might be a risk in that study but
3 we just missed it, and they proceeded to do a risk
4 assessment on that basis.

5 On chart No. 11, the dose response
6 evaluation, in Crump they consistently use, as far as I
7 could tell in going through all of the data, we did not
8 review obviously all the underlying data because they
9 cite hundreds and hundreds of studies, but where they
10 were cited and laid out in their analysis they use
11 always the most sensitive indicator of toxicity for
12 non-cancer effects to establish the no-effect level, as
13 it is called, the basis for deriving the margin of
14 safety.

15 They used the linear no threshold model
16 for carcinogens that I mentioned earlier and even went
17 a little bit beyond the linear model, they used what is
18 called an upper statistical confidence limit on it to
19 put more conservatism into the process. As I said
20 before, for non-carcinogens they assumed a risk.

21 Can I just go back to my graph for one
22 second to give you an idea of how that can be done,
23 2(a). For all of those herbicides where there was
24 animal data, cancer data that were negative or of
25 borderline significance, what Crump did was to say you

1 can calculate where those experiments run out of
2 detection power, roughly around this risk range. So
3 they said, what if there was an excess risk in the
4 animal study right here, we just missed it, and that's
5 possible. So let's assume that risk exists, then you
6 can do the same kind of extrapolation.

7 That's what they did, and I will add to
8 that, that the MOE panel did the very same thing except
9 with this newer set of animal data that was not
10 available to Crump. So both of them looked at that
11 question.

12 I have to emphasize, 2,4-D -- I'm sorry,
13 the MOE panel looked only at 2,4-D, Crump looked at all
14 the other herbicides. So that's what I mean here in
15 chart No. 11 that I was on, third bullet. For those
16 that are not yet shown to be carcinogens but were
17 assumed to be, they used the highest possible risk -
18 and I put that in quotes - that you could derive from
19 the negative data. That has to be kept in mind when
20 you look at the risk results.

21 On the human exposure evaluation, this is
22 probably the most complex part of the Crump report,
23 they considered both workers, those involved in the
24 aerial application, the mixers, the loaders, the
25 pilots, the mechanics, the Department of Natural

1 Resources' personnel who would be nearby and what in
2 the typical risk assessment setting is called the
3 general population. That's the term we use for people
4 other than those involved occupationally, bystanders I
5 guess has been the term used here. It means the same
6 thing, but members of the general population which
7 would include children and older people, et cetera.

8 They considered both of those categories,
9 they looked at every pathway of exposure that could
10 exist. They might have missed some, but I would be
11 hardpressed to point one out. They looked at
12 inhalation of the herbicides from the air, direct skin
13 contact for workers, that's obvious, they also
14 considered direct skin contact of bystanders who might
15 pass through a sprayed area and into contact, say, with
16 plants.

17 They considered ingestion of foods, and I
18 guess I should add here drinking water. They assumed
19 people would drink from streams that might have been
20 affected. Fish, wild game and berries were all
21 considered. They presented both what they call
22 reasonable and worst-case exposures and by that, in
23 going through the analysis where they had data or had
24 to use assumptions, they tried to set out very clearly
25 what they meant by a more typical kind of exposure

1 situation, both for the worker and for members of the
2 general population, and they also considered what might
3 be a worst-case.

4 Let me give you one idea of what they
5 meant by worst-case. I'm not even sure I would go
6 quite this far in the worst-case, but here is the
7 general population exposure that they considered, that
8 an individual would have all of these characteristics,
9 not just one of them or two of them, but all of them,
10 that the individual lives continuously outdoors 24
11 hours a days, that the pesticide -- the only pesticide
12 that individual has contact with is that which came
13 down at the maximum approved usage rate, not other
14 rates, that the total body skin area would be exposed.
15 That's an important determinator of how much gets into
16 the body, how much of the skin area is exposed. They
17 used the assumption of total body area. For their
18 reasonable case, they use half the body area. That's
19 the sort of difference they make.

20 Now, that is an assumption and we don't
21 really know what sort of average body area is exposed,
22 but those were their assumptions. They are probably
23 both quite high. Well, obviously I guess the worst
24 care is fairly high.

25 They used their data on how much of the

1 herbicides go through the skin when they are applied to
2 the skin. There are humans studies on this question,
3 there are animal studies on this question. They give a
4 range of outcomes for the different herbicides and in
5 their worst-case they assumed the high end of every
6 reported value for the amount that goes through the
7 skin.

8 They had -- this person would be eating
9 at two levels of food consumption rates, half kilogram
10 gram per day, for example, of wild game, half kilo, was
11 their estimate of what might be the worst-case and they
12 used a rate that approximates more normal meat
13 consumption based on other data for the more reasonable
14 case. They then took where the more data that shows
15 that the herbicides persist in different environments
16 for different period of time. They used for the
17 worst-case the data shown with the longest persistence
18 after a spraying episode and in the reasonable case
19 they used a more average level.

20 So those are some of the kinds of
21 characteristics that are worked into this analysis and
22 they are all laid out. It is quite an effort to
23 understand that and go through the report, but it is
24 all in there. So, again, those have to be kept in mind
25 when doing this analysis.

1 Now, the last chart here --

2 MADAM CHAIR: Excuse me. Sorry, Dr.

3 Rodricks.

4 DR. RODRICKS: Sorry.

5 MADAM CHAIR: We normally break for lunch
6 at twelve. Is this a good time for you to break or is
7 it...

8 DR. RODRICKS: I will finish this in five
9 minutes I should think, not much longer, unless you
10 have questions. This chart, which is a summary -- it's
11 up to you, five, ten minutes maximum.

12 MADAM CHAIR: I see we will then get into
13 the conclusions on the analysis. I think I would
14 rather hear the last part together.

15 DR. RODRICKS: Okay, that's fine.

16 MADAM CHAIR: Thank you very much. We
17 will be back at 1:30. Thank you.

18 MR. CASTRILLI: Madam Chair, I am
19 wondering if I could simply advise the parties and
20 yourselves of the exhibits I expect I will want this
21 afternoon or be referring to this afternoon and
22 probably tomorrow so that everyone involved can acquire
23 them from their offices, if necessary.

24 Exhibits 714, 715, 716, 717, 754, 789,
25 1233, 1236, and 1237.

1 MR. CASSIDY: Are there any transcript
2 volumes, Mr. Castrilli, that you intend to refer to?

3 MR. CASTRILLI: There actually is one. I
4 actually may not have to refer to it, but it's the one
5 that's referred to, I believe it is 122.

6 Actually, I'm sorry, I can't confirm that
7 at the moment, but I think it is 122.

8 MR. CASSIDY: Are there any other volumes
9 or just that one as a possibility?

10 MR. CASTRILLI: I think it is that one.

11 MR. CASSIDY: All right. Thank you.

12 ---Luncheon recess taken at 12:00 p.m.

13 ---On resuming at 1:30 p.m.

14 MADAM CHAIR: Please be seated.

15 Mr. Cassidy?

16 MR. CASSIDY: Good afternoon, Madam
17 Chair, Mr. Martel.

18 We are prepared to continue with some
19 final comments from Dr. Rodricks. I anticipate we will
20 be approximately another 10 minutes and then Mr.
21 Castrilli has indicated he is prepared to proceed.

22 Dr. Rodricks?

23 DR. RODRICKS: I was just about to
24 present a small piece of the risk characterization
25 section of the Crump report. This table deals with the

1 highest risk that they found. This was individuals
2 involved in mixing and loading pesticides for forest
3 application and this table -- this chart; that is,
4 overhead No. 13, pertains to use of that group.

5 Another very, very important piece of
6 this that must be specified, this is the way Crump
7 presented the data in their summary. This risk
8 pertains to the risk associated with a single spray
9 application. Now, obviously that wouldn't be the total
10 risk, you have to go back into the report to find the
11 total risk and the total risk would be -- I will also
12 report that to you.

13 Because we are dealing with risks which
14 are basically directly proportional to exposure, you
15 can take the risk for a single spray application and
16 answer the question for any other number of
17 applications you might want to look at for, let's say,
18 workers who are involved in spraying and they did that
19 in their report and I will...

20 Anyway, for the single spray application,
21 for 2,4-D, the worst-case risk of cancer that they
22 found - this is what we call assumed because we are
23 assuming that 2,4-D was a carcinogen - was of the order
24 of - I would say less than, because remember also the
25 methodology places an upper bound level on the risk -

1 one in 25 million over a lifetime.

2 There are many different ways that one
3 can look at the question of total risk. In Crump's
4 report they relied upon data from the Department of
5 Natural Resources in the State of Washington on the
6 periods of time people in the pesticide application
7 business hold jobs, how many spray events they might be
8 involved in over a lifetime. In the Crump report,
9 there is a table at the rear of the report that deals
10 with this.

11 I may want to just mention that number,
12 may I? I guess I should have written it down, sorry.
13 I can find it quickly. Table 11-15 of the Crump report
14 contains one estimate of total risks that might be --
15 cancer risks that might be associated with various
16 herbicides for different occupational categories, the
17 pilot, the loader, the mixer loader, the mechanic, the
18 observer.

19 For the worst-case exposure, those risks
20 rise to about one in 100,000 over a lifetime for the
21 loader, for the mechanic and for observers they are
22 still in the order of one to three per million over a
23 lifetime. Now, that would be more specific to the
24 State of Washington.

25 I might add, then, that on this

1 particular issue also, 2,4-D, as I mentioned before,
2 the Ministry of the Environment's expert panel also
3 made a similar kind of analysis for 2,4-D and for
4 workers. Results of that analysis are presented on
5 page 51, Table 8 of the MOE report.

6 This has some advantages over the Crump
7 analysis because, No. 1, it relies upon the more recent
8 animal cancer bioassay data that I mentioned before for
9 their worst-case analysis. Remember, this was a
10 negative study or not sufficient evidence of
11 carcinogenicity, as the panel named it, but they went
12 and performed this analysis on the assumption that it
13 might be a carcinogen.

14 They also have had available, and it is
15 summarized in the MOE report, some more recent data and
16 it is Canada specific -- Ontario specific on
17 occupational exposures to 2,4-D incurred during the
18 application of either aerial application or exposures
19 that are incurred from individuals who are applying
20 2,4-D from backpack, hand-held applications.

21 Their analysis -- and they also looked at
22 the question of the number of days over a lifetime when
23 individuals might be involved in these spray operations
24 in Ontario. They had that information and they had a
25 citation to that. I can only -- I can't verify it is

1 correct, but it is a citation to a department of -- may
2 I just check.

3 Yes, I'm sorry. On page 20 of the MOE
4 report they refer to a survey from DHS, 1987. It was
5 the basis for the number of days in a lifetime that
6 exposures would occur to workers involved in the
7 application of 2,4-D in Canada. So there was a
8 Canadian -- Ontario specific survey.

9 Their risk numbers are in the same range
10 as Crump's. The risk numbers for a full lifetime risk,
11 the highest risk they found was for the backpack
12 sprayer. Those risks are reported in Table 8 of the
13 MOE report, in the order of five to eight per million
14 over a lifetime. Those are the highest risks they
15 found in an occupational setting, and for those
16 involved in aerial application, the mixer, loader
17 risks, as in Crump, were the highest, but in the MOE
18 analysis they were a little under one in a million over
19 a lifetime; somewhat less than Crump reports, but
20 clearly in about the same range. So we have those two
21 analysis which are in fairly close agreement.

22 Just for perspective, in the United
23 States at least, and this comes out in the MOE report
24 for occupational carcinogens, occupational carcinogens
25 in the United States are regulated by the Occupational

1 Safety and Health Administration, and part of the basis
2 for regulation of such carcinogens is a risk assessment
3 of the same type I have talked about here.

4 For workplace exposures, the OSHA, as we
5 call them, have made decisions on about a dozen
6 carcinogens over the last 10 years have not sought to
7 reduce occupational -- lifetime occupational risks for
8 carcinogens below about one per thousand. That's about
9 as low as they get, one per thousand.

10 The MOE report presents a couple of
11 examples, but there are many more to choose from. Now,
12 that is a policy decision to be sure, but these risks
13 are certainly well below - 'these', that is pertaining
14 to 2,4-D under both analysis - are well below what at
15 least has been accepted by OSHA for occupational
16 carcinogens. That may not be the only standard, but
17 that's certainly one.

18 The Crump report deals further with
19 glyphosate and picloram, a similar analysis, yields
20 risks again from a single application very, very much
21 less than one in 25 million for both of these combined.
22 These are lowest risk figures in this chart and, again,
23 if you refer to Table 11-15 of the Crump report where
24 they deal with total lifetime risks in Washington,
25 these rise; that is, glyphosate and picloram, rise to a

1 maximum of about one to five per hundred million over a
2 lifetime. They are extremely small risks.

3 They also deal with margins of safety for
4 reproductive and teratogenic effects and, again,
5 remember this is the worst-case analysis for
6 occupational exposures. I should add that there is
7 also a great deal of information about the general
8 population and all the risks are much lower than these.

9 Margins of safety for reproductive and
10 teratogenic effects, this is the difference between the
11 no-effect level, the maximum dose in which you see no
12 toxicity, and the actual exposure to 2,4-D. They
13 reviewed all the reproductive and teratogenic toxicity
14 data for these three and the other herbicides and found
15 margins of more than 100 for 2,4-D, more than 400 for
16 glyphosate and more than 20,000 for picloram. These
17 vary because these materials have different degrees of
18 toxicity.

19 For other toxic effects, they also review
20 just sort of general toxicity of these materials other
21 than reproductive or teratogenic effects. They found
22 margins of safety from more than five 2,4-D for the
23 worst-case worker, more than 100 for glyphosate and
24 more than 2,000 for picloram.

25 I need to comment on the five a little

1 bit. This is a worker population and a worst-case
2 analysis. Crump characterizes this very, very
3 carefully because they did something a little unusual
4 here. They chose for the no-effect level for -- what I
5 am just calling other toxic effects, it turns out in
6 the case of 2,4-D it will effect the kidney adversely
7 at a sufficiently high dose, so it's just kidney
8 toxicity.

9 They took data from what is called a
10 90-day study where the animals are exposed continuously
11 over 90 days, that is called a subchronic study, to
12 derive a no-effect level for this material and took the
13 margin of safety from that, but now recall we are
14 dealing now with exposures which are highly
15 intermittent and in the human lifetime comprising a
16 much smaller fraction over the lifetime. So there is
17 some additional margin of safety in there beyond the
18 five. It is not really possible to estimate that.

19 I might also add, and this comes out of
20 the MOE report because the no-effect level that
21 Crump -- the toxic effect level that Crump referred to
22 for systemic effects for 2,4-D; that is, for these
23 other toxic effects, came from this 90-day study I
24 referred to. That 90-day study was a study submitted
25 to the Environmental Protection Agency by this Industry

1 Task Force on 2,4-D as a study that was to be used to
2 make decisions about how to design the long-term cancer
3 study they were to do. It is called a screening study,
4 where you try to figure out from results of that study
5 how to dose the animals for a lifetime.

6 What the MOE report points out is that
7 when -- and that was not available to Crump, when that
8 long term, two year exposure study actually took place,
9 there was no effect on the kidney seen at the dose
10 where you saw something in the 90-day study. No effect
11 whatsoever.

12 The MOE doesn't try to explain that,
13 nobody has tried to explain that very much except so
14 say that - and this is in the MOE report - that in all
15 studies there are effects you will see in various
16 organs that occur spontaneously in animals and you try
17 to separate those from effects due to the chemical. In
18 this case, we are not sure why we had the observation
19 in 90 days of something going on in the kidney and no
20 such effect over -- we the exposure was longer at the
21 same dose. So there is a little uncertainty there
22 about where that no-effect level is, but the long term
23 study should give us some comfort.

24 The reasonable exposure scenarios, the
25 more typical exposures, of course the risks are

1 smaller, roughly on average 80 times smaller than the
2 worst-case cancer risks and safety margins are about
3 four times larger for the reasonable exposure
4 situation. Again, these are for workers, the general
5 population much less.

6 I will close then just to mention some
7 other characteristics of the Crump analysis. We were
8 asked to comment on the general procedures, the quality
9 of the report and the group. Dr. Crump is one of
10 the -- as I say, is an internationally recognized
11 expert in chemical risk assessment. That is kind of an
12 understatement.

13 He is one of the people whose
14 publications during the late 60s and early 70s really
15 gave rise to much of what we do in risk assessment.
16 So he is really quite widely acknowledged as an expert
17 in the area.

18 The methodology used conforms in all
19 respects to that recognized, at least in the United
20 States, as an appropriate way to evaluate risks.

21 Q. I understand that you wish to file
22 copies of the guidelines that are used in the United
23 States for assessing carcinogen risk?

24 A. Yes. The Environmental Protection
25 Agency has actually published from time to time reviews

1 or what they call guidelines on how they go about the
2 cancer risk assessment process.

3 MR. CASSIDY: I have a copy of those to
4 provide to the Board, Madam Chair, as the next exhibit
5 which would be Exhibit 1246, entitled Guidelines for
6 Carcinogen Risk Assessment, dated September 24, 1986
7 published by the U.S. EPA.

8 ---EXHIBIT NO. 1246: Document entitled Guidelines for
9 Carcinogen Risk Assessment, dated
10 September 24, 1986 published by
the U.S. EPA.

11 DR. RODRICKS: With respect to the
12 worst-case analysis, bullet three on this last chart,
13 we couldn't find anything in their analysis that I
14 wouldn't consider worst-case for their worst-case
15 scenario. It seemed to be appropriately conservative,
16 in some cases more conservative than I would have
17 carried out. More conservative in procedures that were
18 adopted than I would have adopted.

19 There is no clear single definition of
20 what you mean by a worst-case analysis and how you put
21 together the data to reach a conclusion about
22 worst-case. I showed you some examples from the
23 report. They seemed to be a bit extreme, but given the
24 outcome of the assessment, I don't think that's
25 problematic, but this seems to be -- excessively

1 pessimistic, as I've said here.

2 One of the reasons you do a worst-case
3 analysis and risk assessment is that it is generally
4 easier to do, requires less data and information
5 because you could substitute some pretty extreme
6 assumptions like the hundred per cent body exposed sort
7 of assumption, which I guess would have to be read as
8 as a worst-case assumption. You can do that sort of
9 thing in a worst-case analysis without collecting for
10 some kinds of information a lot of data. So this is
11 not an uncommon practice.

12 If the result of that very worst-case
13 analysis yields risks which are extremely low, that's
14 important information because it says -- gives you a
15 couple of clues, the most important one of which it
16 tells you whether it is really worthwhile proceeding to
17 get additional information. If the risks under a sort
18 of very worst-case assumption seemed to be very, very
19 low, then that raises a question whether it is really
20 valuable to learn something new. So that's one of the
21 kinds of uses.

22 The only qualification on its
23 comprehensiveness, I think it's reliable, is that it
24 was done in -- completed in '86 and would not have
25 covered any literature like the ITF study that I

1 mentioned since the time it was done, but in other
2 respects it seems quite comprehensive to us.

3 I think that's all I have.

4 MR. CASSIDY: That completes the evidence
5 of this panel in-chief, Madam Chair.

6 MADAM CHAIR: Thank you, Mr. Cassidy.

7 Mr. Castrilli?

8 DR. RACHMAN: Madam Chair, while we are
9 making this change can I be excused for a moment.

10 MADAM CHAIR: Certainly.

11 MR. CASTRILLI: Madam Chair, you can tell
12 someone much taller than I was at the podium a moment
13 ago. I will just be one more minute.

14 CROSS-EXAMINATION BY MR. CASTRILLI:

15 MR. CASTRILLI: Q. Dr. Rachman, can we
16 begin with you at page 14 of your evidence. You are
17 discussing there the special review process under
18 FIFRA. I just want to clarify one comparatively minor
19 point.

20 In your summary on those pages with
21 respect to the special review process, my understanding
22 is that that process includes a consideration of
23 whether a pesticide that is already registered on the
24 basis that it will not cause unreasonable adverse
25 effects on human health may in fact be causing such

1 effects?

2 DR. RACHMAN: A. That's correct. If I
3 understand your question correctly, a pesticide that
4 has already been registered and, therefore, which has
5 been found to satisfy the no adverse effect criterion,
6 may subsequently go into special review.

7 That scenario would apply if additional
8 information were involved that changed the scientific
9 picture.

10 Q. Fine, thank you for that
11 clarification. And I understood your testimony on this
12 issue as well, that the result -- or the results of a
13 special review could include, for example, continued
14 unmodified registration, that's one option?

15 A. That is an option, yes.

16 Q. And a further possible option might
17 be use restrictions or label modifications?

18 A. Yes. The point I was trying to make
19 is that the agency might take a variety of actions.
20 They would choose the one that would be most
21 appropriate to mitigate the risk that had been
22 identified as significant in the special review.

23 Q. And a further result of the special
24 review process might be cancellation or suspension of
25 the registration; is that correct?

1 A. It could be a proposal from the
2 agency for cancellation or suspension and then, of
3 course, those procedures would have to be played out
4 under the provisions of the law and the regulations.

5 Q. Okay, thank you. Now, I just wanted
6 to refer you as well, Dr. Rachman, to page 20 of your
7 evidence. Now, I understand that -- I'm sorry, Dr.
8 Rachman, we are in particular referring to your
9 discussion on that page regarding the special review
10 process as it applies to 2,4-D, so we are looking at
11 the bottom of that page.

12 As I understood your testimony this
13 morning, the information contained on page 20 and over
14 to page 22 really now has to be considered in light of
15 what is now Exhibit 1242, which is the--

16 A. The latest, yes.

17 Q. --Federal Register for October 13,
18 1989; is that right?

19 A. That's correct.

20 Q. And as I understand Exhibit 1242,
21 what the agency is saying is that it is postponing its
22 decision not to initiate a special review of 2,4-D; is
23 that correct?

24 A. That's my understand of that, yes.

25 Q. And as I understand it, among the

1 reasons given by the agency for that decision to
2 postpone a decision not to initiate a special review
3 include a number of ongoing epidemiological studies; is
4 that correct?

5 A. Yes, that's my understanding.

6 Q. And that's reflected, for example, in
7 the summary that you, I believe, read into the record
8 at page 42,032 of Exhibit 1242? It is the left-hand
9 column of that exhibit. Do you not have a copy of the
10 exhibit?

11 A. I am getting my numbers confused
12 here.

13 Q. All right.

14 A. Just one moment. We have a copy of
15 that exhibit here.

16 MADAM CHAIR: Do you want to repeat that,
17 Mr. Castrilli?

18 MR. CASTRILLI: Madam Chair, we are
19 referring to Exhibit 1242 which the U.S. Federal
20 Register -- or I should say U.S. EPA Federal Register
21 document dated October 13, 1989 and we are referring to
22 the summary paragraph which is called Summary.

23 MADAM CHAIR: Thank you.

24 MR. CASTRILLI: The first column on page
25 43,032.

1 DR. RACHMAN: Okay. The summary
2 paragraph being the first full paragraph?

3 MR. CASTRILLI: Q. Yes. Actually, it is
4 the paragraph called Summary as it happens.

5 DR. RACHMAN: A. Mr. Castrilli, I'm
6 terribly sorry, could you please repeat this question?
7 I have just gotten lost.

8 Q. Yes. As I understand it, the
9 reason -- among the reasons the agency has given for a
10 decision to postpone its decision not to initiate a
11 special review of 2,4-D includes the fact that there
12 are a number of epidemiological studies which are
13 ongoing and which the agency wishes to consider before
14 it makes a final determination; is that correct?

15 A. Yes.

16 Q. Thank you.

17 MADAM CHAIR: Excuse me, Dr. Rachman.
18 Are they only the two studies that we heard about in
19 Nebraska and in Iowa?

20 DR. RACHMAN: Those are the two studies
21 that are specifically mentioned in this notice, Madam
22 Chair.

23 If you will look at page 42,034, the
24 left-hand column, the top paragraph says Forthcoming
25 Data, and they describe the studies in that paragraph.

1 One from eastern Nebraska, the other from Iowa and
2 Minnesota, both NCI studies in progress.

3 MADAM CHAIR: And then the final sentence
4 says:

5 "Other epidemiological studies which may
6 provide some information about 2,4-D are
7 being performed or planned by NCI."

8 DR. RACHMAN: Yes. I have no information
9 about what those studies might be.

10 MR. CASTRILLI: Q. And just bear with me
11 for one moment, Dr. Rachman. The current status,
12 therefore, of 2,4-D in the U.S. EPA regulatory process
13 is that no final decision has been made on whether to
14 initiate a special review; is that correct?

15 DR. RACHMAN: A. Yes. I would agree
16 with that. Whether that constitutes regulatory status,
17 I think it's a legal question which I really can't
18 answer. I mean, no decision has been made as of this
19 time that a special review is warranted and it's going
20 to stay that way until this additional information is
21 reviewed.

22 Q. Dr. Rachman, we are still at page --
23 actually, let's go to your summary or your -- what do
24 you call it, your executive summary?

25 This would be (iv), paragraph 12.

1 MR. CASTRILLI: Madam Chair, in case that
2 was not clear, it's page (iv), paragraph 12, a portion
3 of the executive summary for this exhibit which is
4 Exhibit 1239.

5 Q. Dr. Rachman, let me just read the
6 paragraph into the record that I am referring to.

7 "The EPA has not identified any risks
8 with respect to forestry use in the
9 United States of the chemical pesticides
10 approved for forestry in Ontario and has
11 not imposed, with respect to these
12 pesticides, any form of risk mitigation
13 requirement related to human health
14 effects from forestry uses."

15 Dr. Rachman, I gather, therefore, by
16 deduction that that is the situation, in your opinion,
17 with respect to 2,4-D as well?

18 DR. RACHMAN: A. Yes, that's correct.
19 My understanding is that no risks have been identified
20 from the forestry uses of 2,4-D and no risk mitigation
21 measures have, therefore, been imposed.

22 Q. Dr. Rachman, can you confirm for me
23 that the U.S. EPA is not the only federal agency in the
24 United States that make decisions with respect to the
25 health and environmental risk posed by herbicides such

1 as 2,4-D?

2 A. No, in fact I cannot confirm that,
3 Mr. Castrilli. The EPA has the responsibility for the
4 federal registration of the pesticide and that
5 registration involves the determination of whether or
6 not unreasonable adverse effects are likely to occur
7 under the proposed conditions of use, as I explained in
8 my testimony.

9 Now, there are other agencies, both
10 federal and state and even local, that make decisions
11 in the United States with respect to the use of various
12 pesticides and the conditions of use of various
13 pesticides. They may do independent reviews of data,
14 but their decisions do not affect the federal
15 registration status in any way.

16 Q. Let me be more specific. Are there
17 other federal agencies in the United States that make
18 decisions with respect to environmental health risks,
19 if I can use that term, posed by herbicides such as
20 forestry -- excuse me, such as 2,4-D for forestry use?

21 A. The United States Forest Service,
22 which is part of the U.S. Department of Agriculture,
23 does environmental impact analyses of the proposed
24 forestry uses in the United States.

25 As I'm not and expert in this area, my

1 understanding is that under the requirements of the
2 National Environmental Policy Act major undertakings
3 that are proposed by government agencies -- the
4 environmental impacts of those proposed undertakings
5 have to be evaluated, very similar to the proceeding
6 that we are taking part in here.

7 In that sort of proceeding, the forest
8 service will review and evaluate evidence relating to
9 environmental health risk of pesticides. I will just
10 leave it at that.

11 Q. Thank you. Do you have Exhibit 1237
12 before you? That would be the Ozark -- it is the very
13 last document on your desk.

14 A. The big one?

15 Q. Yes. Not quite the Toronto phone
16 directory but I guess it would do in a pinch.

17 A. Yes.

18 Q. Dr. Rachman, are you aware that the
19 various regions -- sorry. Let me withdraw that
20 statement since that it is not true in the context of
21 the question I was about to ask you.

22 Would you agree with me that there is at
23 least one forest region in the United States U.S.
24 Forest Service System that has made a decision about
25 whether to permit 2,4-D use?

1 A. I have not --

2 Q. For forestry purposes?

3 A. I have not had an opportunity to
4 review this document, Mr. Castrilli, so I cannot
5 confirm that for you.

6 Q. Let me refer you to one or two pages
7 very briefly from that exhibit. Looking at (ii) --
8 sorry, the chapters are divided by large Roman
9 numerals, so we are looking initially (II)-(LV), That
10 would be the page number. We are looking at Item 2 on
11 that page, which states:

12 "Only herbicide formulations (active
13 and inert ingredients) and additives
14 registered by EPA..."

15 Just stopping there, Dr. Rachman. Your
16 understanding would be that that is under FIFRA; is
17 that correct?

18 A. That's correct.

19 Q. And continuing in the sentence:

20 "...and approved by the forest service
21 for use on national forests are applied."

22 Just stopping there. Would you confirm
23 that that basically describes what you have just
24 indicated to the Board a moment ago, that certain
25 forest services as a result of the NEPA process are in

1 a position to make decisions about what herbicides are
2 used in certain national forests. Is that your
3 understanding?

4 A. Yes, that would be my understanding.
5 I would like to point out, though, that the decisions
6 of whether or not or when to use a particular pest
7 management option fall in the realm of risk management
8 that we discussed where more is taken into
9 consideration than simply the scientific findings of
10 risk.

11 There are policy issues at stake here and
12 I'm sure that part of the decision that the forest
13 service makes in deciding to use any particular
14 pesticide takes into account public opinion and a lot
15 of other things.

16 MR. MARTEL: You are saying it is not
17 necessarily all due to the toxicity, if I can use that
18 term, of the substance that they are banning, in other
19 words?

20 DR. RACHMAN: Yes.

21 MR. MARTEL: There are other motives.

22 DR. RACHMAN: That would be my
23 understanding based on similar documents that I have
24 seen in the past.

25 MR. CASSIDY: Mr. Martel, I wonder if I

1 might ask you, when you are speaking if you could
2 perhaps more your microphone just a little bit closer.
3 Thank you very much.

4 MR. CASTRILLI: And if the reporter
5 cannot hear me, please let me know.

6 Q. Dr. Rachman, can I now ask you to
7 turn to page -- this would now be (xii) in Exhibit
8 1237. A unique number system. Actually, it's not such
9 a small number but it is (xii).

10 Dr. Rachman, this is under a general
11 heading Environmental Consequences and looking at the
12 first subheading on the page entitled health -- excuse
13 me, entitled Human Health and safety, I will read the
14 paragraph into the record and I would like to ask you a
15 question about this paragraph.

16 "All herbicides and additives
17 investigated provide ample margins of
18 safety for the public when applied using
19 typical rates and methods. However,
20 because 2,4-D, 2,4-DP..." and two other
21 herbicides, some of which I may not be able to
22 pronounce, but in any event they are not used in this
23 hearing,

24 "...have lower margins..." would never
25 be used in this hearing and are not proposed to be used

1 in the area of the undertaking,

2 "...have lower margins of safety or pose
3 possible environmental risks they were
4 not considered for use in the
5 Ozark/Ouachita Mountains area."

6 I will just read the last sentence:

7 "In general, worker exposure is reduced
8 by aerial application."

9 Were you aware, Dr. Rachman, that this
10 particular U.S. forest service was not -- or is not
11 going to permit the use of 2,4-D in the Ozarks because
12 of lower margins of safety or possible environmental
13 risks?

14 A. No, I was not aware of that.

15 Q. Dr. Rachman, can I now refer you to
16 Exhibit 1236. This would be a document, it look likes
17 that. It is entitled Record of Decision, USDA Forest
18 Service. It is dated March 5, 1990. We are looking at
19 page 7. At page 7 we are looking at the middle of the
20 page, the paragraph 1 that's in brackets that begins:
21 "Only herbicides..."

22 Do you have the paragraph?

23 A. (nodding affirmatively)

24 Q. I will read that into the record.

25 "Only herbicides with least envir --

1 excuse me.

2 "Only herbicides with least health
3 and environmental risks may be applied
4 and only at most lowest effective rates."

5 There is a reference to the final EIS that I referred
6 you to a moment ago.

7 "The herbicides that may be used are..."
8 and they are identified there. They include for the
9 purpose of this undertaking:

10 "...herbicides such as glyphosate,
11 hexazinone, picloram (only products
12 formulated without 2,4-D)..."

13 It goes on to identify several other
14 herbicides that are going to be permitted to be used in
15 that forest region and also identifies a number of what
16 appear to be inert ingredients that will be permitted.

17 Dr. Rachman, were you aware that in
18 addition to not permitting 2,4-D use in its own right,
19 that the regional forester for the U.S. Forest Service
20 in the Ozarks has decided not to permit the use of
21 picloram if it is formulated with 2,4-D?

22 A. No, I was not aware of that.

23 Q. Can I refer you now to your evidence
24 again, Exhibit 1239, and we will be looking at page 53.

25 Actually, Dr. Rodricks, to be fair to

1 you, I think it actually contains a better summary, we
2 might also look at page (viii) of your evidence,
3 paragraph 22.

4 DR. RODRICKS: A. Paragraph 22?

5 Q. Yes, that's correct.

6 Dr. Rachman -- Dr. Rodricks, excuse me,
7 on that page you indicate that there are several
8 uncertainties attached to the Kansas study and its
9 results and you summarize the uncertainties in
10 paragraph 22 and you expand upon them in pages 50 to 56
11 of your evidence. I believe you restate the concern
12 with respect to uncertainties at page 53 of your
13 evidence.

14 Just as a general proposition, Dr.
15 Rodricks, would it be fair to say that in general there
16 are uncertainties associated with every epidemiology
17 study?

18 A. I think that's probably a pretty good
19 generalization, yes.

20 Q. And in particular with respect to the
21 Kansas study, can you confirm for me that it was
22 reviewed by both the EPA and outside consulting
23 epidemiologists and was found to be well designed and
24 well executed?

25 A. I know of no serious criticism of the

1 design or execution of that study.

2 Q. Just to -- I'm sorry, I didn't mean
3 to cut you off.

4 A. Are you referring to specific
5 statements about design?

6 Q. Yes. Let's look at Exhibit 1242?

7 A. 1242.

8 Q. That's the exhibit you filed this
9 morning. We are looking at page 42,033. Dr. Rodricks,
10 we are looking at the first column under the heading
11 Epidemiologic Evidence and we are looking at the
12 last -- next to last sentence on the page.

13 A. Yes, I see that.

14 Q. And the statement reads:

15 "The study..." and this is of course a
16 reference to the Kansas study,

17 "...was reviewed by EPA and consultant
18 epidemiologists and found to be well
19 designed and executed."

20 Would you agree with that assessment, Dr.
21 Rodricks?

22 A. Yes, I think -- yes, generally.

23 Q. Just looking at the last sentence on
24 that page, let me read that that into the record.

25 "However, after an indepth evaluation,

1 EPA concluded that the study did not
2 provide sufficient evidence of a link
3 between 2,4-D and NHL..."

4 Dr. Rodricks, you realize that in Canada
5 NHL is the National Hockey League.

6 A. In the U.S. as well.

7 Q. That's true.

8 "...to pursue regulatory action at that
9 time."

10 Now, just stopping there. I would like
11 your opinion on the sentence and what its import is.

12 Would it be -- is your interpretation of
13 that -- or is a fair interpretation of that sentence
14 that what EPA is stating is that they are acknowledging
15 that there are limits to what one can expect from any
16 single epidemiology study? Is that a fair
17 interpretation of that sentence?

18 A. Well, it's a little more specific
19 than that. I mean, I read it to say that they have
20 looked at it deeply and the study by itself does not
21 establish a link, in this case, between 2,4-D exposure
22 and a risk of non-Hodgkin's lymphoma sufficient to
23 pursue regulatory action.

24 So it is a little more specific, but you
25 have the general intent of it.

1 Q. All right, thank you. If I might,
2 Dr. Rodricks, I would like to put another general
3 proposition to you. By some time tomorrow I will get
4 down to specifics.

5 MR. CASSIDY: You have until Friday.

6 MR. CASTRILLI: That's my understanding,
7 yes.

8 Q. As a general proposition, Dr.
9 Rodricks, would it be fair to say that evidence of
10 carcinogenic activity of a chemical agent can be
11 obtained from epidemiological studies when evaluation
12 of the observation shows that the chemical agent causes
13 an increased incidence of neoplasms?

14 DR. RODRICKS: A. Well, the critical
15 word there is 'causes'. If you can establish
16 causation, as I discussed in my presentation this
17 morning, that is not an easy thing to do with
18 epidemiologic methodologies, but if you can establish
19 causation and if you have reached that point through a
20 series of studies where you see excesses of a certain,
21 as you put it, neoplasm or cancer, then, yes, it can
22 happen.

23 As I said, there are approximately 30
24 chemicals or mixtures of chemicals where such causal
25 links have been established, not including 2,4-D.

1 Did I get your question right?

2 Q. I think you had the gist of the
3 question. Let me pursue this a moment with you for a
4 moment. Would it also be fair to say that evidence of
5 carcinogenic activity of a chemical can be obtained
6 from such human studies when the evaluation of the
7 observations also shows that the agent causes a
8 decrease in their latency period?

9 A. Well, that is one piece of evidence.
10 I doubt if that -- if you mean from a single study an
11 observation of a decreased latency period associated
12 with an exposure, decreased time from first exposure to
13 the finding of the tumour, in a single study as sole
14 evidence of causation that would be highly unlikely,
15 but it is one of several things you'd look at.

16 I didn't mention latency in my five
17 criteria this morning, but it is subsumed under the
18 dose response relationship.

19 Q. I'm sorry, you are competing with a
20 fire engine. I missed the last part of that.

21 A. I didn't specifically -- if you
22 remember my five criteria from the overhead this
23 morning for evaluating evidence of causation, one of
24 those was increasing risk with increasing exposure.

25 I perhaps should have added, and it is

1 subsumed under this, that if you also see decreased
2 latency; that is, decreased time from first exposure to
3 the first observation of a case with increasing
4 exposure, that is a similar piece of confirmatory
5 evidence.

6 But if your question was, if you saw that
7 in a single study and that was the only evidence you
8 had to link an exposure to a disease, that would not be
9 sufficient by itself. It is just one of several things
10 one would want to see.

11 Q. I perhaps misled you as to the focus.
12 I wasn't in that particular question focusing on one
13 study per se. I think I used the plural, or I hope I
14 use the plural.

15 Your would your answer change if you now
16 understand the question to relate to more than one
17 study?

18 A. Could you do the question again,
19 please? The question was...

20 Q. Sure. Is evidence of carcinogenic
21 activity -- or can evidence of a carcinogenic activity
22 of a chemical be obtained from epidemiologic studies,
23 plural, when evaluation of the observation shows - I
24 think I asked you initially - an increase of incidence
25 of neoplasms; and, secondly, a decrease in their

1 latency period?

2 A. If that were found in different
3 studies, preferably done in different populations with
4 different study methods consistently, that would be
5 quite powerful evidence, yes.

6 Q. All right, thank you. Would it be a
7 fair statement that -- or is it a statement you can
8 agree with, that clinical signs of cancer can be
9 delayed for a long time after initial exposure to a
10 carcinogen or to carcinogens?

11 A. Yes, that is the latency period,
12 so-called, and it can very long for carcinogens,
13 perhaps up to 40 years in some cases from the time of
14 initial exposure until the appearance of the disease.

15 Q. And would it be fair to say that the
16 latency period can be from approximately five years to
17 40 years from initial exposure until the disease
18 appears?

19 A. There are some agents where a latency
20 as short as five years has appeared. I would guess the
21 average to be in the 20- to 25-year range and there are
22 some that go up to 40 years. Some asbestos producing
23 cancers, for example, go up to 40 years. So there is
24 quite a range.

25 Q. Would you also agree with the

1 proposition that evaluation of epidemiologic studies
2 requires a knowledge of the smallest possible increase
3 in tumor incidence detectable under the conditions of
4 the study or studies?

5 A. I think your question has to do with
6 the power of the study to find an effect. I mentioned
7 in my - if you remember - graph this morning where I
8 showed that even in animal studies and also in
9 epidemiologic studies there is just a limit to the rate
10 of disease that can be detected. It is a function of a
11 number of factors, most importantly the size of the
12 population which you are able to study.

13 So what you are asking is whether one
14 could estimate the power of a study to detect an
15 effect; that is, if a study is negative, what risk
16 could it have missed, and there are -- sometimes from
17 epidemiology studies there are data available that
18 allow you do that, although it is not an easy thing to
19 do when you do not have exposure information,
20 quantitative exposure information, but I've done that
21 sort of calculation myself for some agents, but whether
22 you can do it depends on the kinds of information you
23 have available from the epidemiology study. It is not
24 always possible.

25 Q. Okay. I gathered from your answer to

1 my question that this type of information is of
2 critical importance in the evaluation of apparently
3 negative studies?

4 A. Well, you should try to do it, I
5 agree with that, to see whether the negative study
6 would have -- how much of an effect a negative study
7 could possibly have missed, yes. But I also emphasize
8 thta it is not always easy to do if you don't have the
9 data.

10 Q. Dr. Rodricks, we are still keeping
11 this at a comparatively general level. Would it be
12 fair to say that substances widely distributed in
13 commerce or the environment are difficult to study by
14 epidemiologic methods in part because it is often
15 impossible to identify unexposed groups as controls?

16 A. Your premise was substances that are
17 very widely distributed in the environment?

18 Q. Yes.

19 A. Well, you may be able to discover
20 occupational exposures, this is typically the case.
21 They are much more intense than general environmental
22 exposures and even though there is -- even though you
23 can't say that the exposure in the general population
24 is zero, it still may be small relative to, let's say,
25 an occupational setting.

1 So there may be opportunities if you can
2 identify the appropriate occupational group to examine.
3 So I think it very much depends on whether you have
4 that opportunity.

5 Q. Let me try this one again and use a
6 different example. This is again with respect to
7 substances that are widely distributed in the
8 environment. We will forget about commerce for the
9 moment.

10 Do I understand your testimony to be --
11 or indicate if you can agree with me that for
12 substances of that type it's particularly difficult to
13 study them by epidemiologic methods or to separate --
14 because it is impossible to separate out groups with
15 high and low exposure?

16 A. And my answer was that you might be
17 able to find occupational groups, people who are, say,
18 in the manufacturing of the material who have very much
19 higher exposures than the general proposition
20 population.

21 So you still may have the opportunity to
22 study them, but if there is no such group or you cannot
23 distinguish groups based on significant differences in
24 exposure, then it's probably a fairly futile effort to
25 try to investigate that.

1 Q. And I think you've already indicated
2 that -- or maybe you haven't, let me just put the
3 proposition to you.

4 Is it a fair statement to say that the
5 problem of adequate controls is further compounded by
6 the long latency period for cancer?

7 A. Well, it's made difficult by several
8 factors, that is one of them, to identify populations
9 that may act as appropriate controls. There are
10 several things that may make that more difficult. I
11 agree that that is one of them.

12 Q. And that could include, for example,
13 multiple opportunities for exposure to other possible
14 substances that might be carcinogenic?

15 A. You mean is that a problem -- does
16 that create difficulties in identifying a control
17 population?

18 Q. Yes.

19 A. Well, that creates lots of
20 difficulties for either -- for both the control and the
21 study population. But generally, yes, I would agree
22 with that.

23 Q. Thank you. Would it also be fair to
24 say that the effects of other exposures on rates of
25 cancer are rarely known and sometimes can have more

1 than an additive effect?

2 A. You are talking now generally still?

3 Q. Yes.

4 A. And I guess your question is, if you
5 are studing -- I guess I don't understand your
6 question. There seemed to be two.

7 Q. If you are focusing on one substance
8 but you also have the possibility of multiple exposures
9 to others, does that affect your understanding of the
10 rate of cancer and, in particular, does it affect
11 the -- whether in fact the effect you are observing can
12 be more than additive?

13 A. I don't think so. In a typical study
14 of an occupational group that's exposed to a chemical,
15 if you are interested in that one chemical, it is
16 almost always the case that those same workers are
17 exposed to other chemicals because lots -- most of,
18 say, manufacturing involves multiple chemical
19 exposures.

20 You try to match those against some
21 control group that is identical in all respects except
22 they don't have exposure to the chemical you are
23 interested in or they have a very much reduced
24 exposure. That's a very hard thing to accomplish and
25 that's the reason these studies are not controlled.

1 Now, you do that the best you can and you
2 may observe an excess of, say, cancer in that worker
3 group. Now, what you can say about the excess then is
4 limited by the fact that they had multiple chemical
5 exposures. So you could say, well there seems to be an
6 excess of cancer in this occupation, but we are not
7 sure of what it is due to. That would be very hard to
8 figure out.

9 Now, whether the excess you see involves
10 some additive or multiple effect of mixtures of
11 chemicals, you can't tell from that kind of study.
12 That requires much more sophisticated investigation.
13 There are such cases, but you can't tell it from a
14 simple single epidemiologic study.

15 Q. Well, I am not particularly
16 interested in whether we are trying to establish it
17 from one study only, let's keep it plural.

18 A. All right.

19 Q. You raised an issue I just wanted to
20 follow up on. If an effect is more than additive it is
21 synergistic; is that correct?

22 A. That's the general term that is used,
23 yes.

24 Q. And perhaps just for the record,
25 could you just indicate to the Board what synergism is?

1 A. Agent A causes a risk "x" at a given
2 level, agent B by itself causes risk "y". An additive
3 relationship, if you put agent A and agent B together
4 so both exposures occur, the total risk if they are
5 additive is "x" plus "y".

6 There are some cases where when you put A
7 and B together the risk is more than "x" plus "y".
8 When it is more it is called a synergistic effect. If
9 it's just "x" plus "y", that's an additive effect.
10 There is also antagonism which is less than "x" plus
11 "y".

12 Q. So with respect to -- I'm sorry?

13 A. Okay.

14 Q. So with respect to synergism, then,
15 the effect of two agents could be greater than -- when
16 put together could be greater than the effect of either
17 of them operating separately?

18 A. Yes. There is one very good example
19 of that in cancer and that is the combined effect of
20 asbestos exposure and smoking.

21 Q. Okay. Dr. Rodricks, I wonder if we
22 could just summarize what I think is the gist of what
23 you have told us so far arising from the questions that
24 I have asked with respect to cancer.

25 The factors that can contribute to the

1 insensitivity of human or epidemiologic studies can
2 include, as I understand your testimony --

3 A. You said the word insensitivity?

4 Q. Yes.

5 A. Okay.

6 Q. Can include the long latency period
7 between exposure to an agent and the onset of cancer
8 and, as we discussed, it could be between 5 and 40
9 years?

10 A. Yes.

11 Q. The high background rate for cancer?

12 A. Generally -- we didn't cover that,
13 but if you are trying to deal with cancers that have a
14 very high prevalence in the population, that reduces
15 the sensitivity, yes.

16 Q. And, thirdly, exposure to several
17 carcinogens over one's lifetime?

18 A. I'm not sure that reduces
19 sensitivity, that complicates the problem of finding a
20 causal relationship to a specific agent. I'm not quite
21 sure that has to do with sensitivity.

22 Q. All right, that's fine. Now, I want
23 to turn to your discussion of the Kansas study, and I
24 wonder if I could refer you initially to page 53.

25 I believe we are looking at the top of

1 the page and you indicate there that:

2 "An odds ratio..." the acronym is OR,
3 "...of 1.0 signifies no difference
4 between cases and controls for the
5 studied exposure."

6 A. Yes.

7 Q. And in the -- sorry, let me just go
8 through a couple of these portions of this page before
9 I get to my first question.

10 In the next paragraph, paragraph 2 on
11 page 53, you are discussing your opinion that the range
12 of variation, which is 1.9 to 19.5, in the odds ratio
13 estimate of risk of contracting non-Hodgkin's lymphoma
14 is so great that the estimate itself, because it is
15 based on very few cases, must be considered unstable.
16 Is that your testimony still?

17 A. Yes.

18 Q. And just so I understand what you
19 mean by the word 'unstable', Dr. Rodricks, do you mean
20 that the range of ORs could change with the addition of
21 extra cases?

22 A. It means that we are dealing with so
23 few, such a small number of cases here that one more or
24 less could change that greatly. It's very, very
25 sensitive to a small change. That's all it means.

1 Q. Would you agree with me, Dr.
2 Rodricks, that the chance of an additional case
3 dropping the range to 1.0 is equal to the chance that
4 one extra case could raise the OR above 19.5?

5 A. I'm sorry, you are assuming one more
6 case is found in this cohort?

7 Q. Let's assume that.

8 A. And then the question was, if you did
9 find one more case the odds ratio would go greatly
10 above six?

11 Q. Sorry, no, I meant the range. Let me
12 restate the sentence -- restate the question.

13 A. Okay.

14 Q. Would you agree with me that the
15 chance of an additional case dropping the range to 1.0
16 is equal to the chance that one extra case could raise
17 the range above 19.5?

18 A. A chance of one more case dropping
19 the range to one?

20 Q. Yes.

21 A. Assuming that the controls have no
22 more cases. The bottom end of the range is not going
23 to go -- if that happens it's not going toward one, it
24 is going to go the other way.

25 Q. It's going to get higher?

1 A. Yes. In other words, there is a
2 greater likelihood of -- it is a stronger association
3 if you add one more case, and I can't calculate that
4 here.

5 Q. Would you agree with me that it is
6 highly unlikely that with additional cases the range is
7 going to centre around 1.0?

8 A. Well, this observation was -- for
9 these cases the odds ratio was already six. So if your
10 assumption is we are going to find more cases and the
11 control rate is not going to change, the odds ratio is
12 going to increase and increase and confidence interval
13 around that is going to shift accordingly and probably
14 become narrower in fact because you've got more cases.
15 It is not going to go down to zero.

16 Q. The data from the Kansas study, Dr.
17 Rodricks, says that there is only a 5 per cent chance
18 that the OR is less than 1.0; isn't that right?

19 A. For this particular finding it says
20 that there is only a 5 per cent chance that it is less
21 than 1.9.

22 Q. All right. There is a 95 per cent
23 confidence that the OR is between 1.9 and 19.5; is that
24 right?

25 A. That's correct.

1 Q. And you indicate that the range is
2 above an OR of 1.0; is that right?

3 A. Yes, that's another way of saying
4 this is a statistically significant odds ratio.

5 If the -- you will see in a lot of the
6 epidemiology studies that the odds ratio is reported
7 within the confidence limits and if the lower
8 confidence limit is above one, that generally means we
9 are talking about a statistically significant
10 association.

11 Q. Let's continue with page 2 -- sorry,
12 page 53 and now look at paragraph -- or Item 2 at the
13 bottom of that page, and actually the paragraph goes on
14 to page 54. Let me just read the paragraph into the
15 record so we understand the context.

16 "The chi-square test was employed to
17 evaluate whether or not a trend for
18 an increasing frequency of herbicide use
19 existed. A significant trend was found.
20 This trend is accounted for primarily by
21 the significant excess risk associated
22 with the highest frequency of use
23 category when this experience is compared
24 with that of nonfarmers. There was no
25 difference in the ORs for the first three

1 categories of days of use per year, but
2 the two highest categories showed some
3 elevations. This suggests that while a
4 trend may exist, this may be open to
5 question. It is questionable whether the
6 establishment of a trend based primarily
7 upon an excess in one data point, is
8 appropriate and reliable..."

9 Dr. Rodricks, just so I understand the
10 chi-square test, if the Kansas study passed this test,
11 would you agree with me that by definition a
12 significant trend was established for increasing
13 frequency of herbicide use?

14 A. Well, we are talking about a trend
15 here, we are talking about an increase in the rate,
16 number of cases, if you like, of non-Hodgkin's lymphoma
17 as a function of frequency of use of herbicides, and
18 there certainly was an excess in the high exposure
19 group, the one defined a little bit earlier with the
20 odds ratio of six.

21 You see, a trend refers to a relationship
22 between exposure and the odds ratio. That is, as we
23 talked about earlier, likely to be the phenomenon of
24 increasing odds ratio with increasing exposure. The
25 trend refers to that sort of dose response

1 relationship, not to a single point.

2 And this is not a specific criticism
3 here, but simply to point out that finding -- that the
4 single elevation where others were not elevated is not
5 strong evidence of a trend in the data. You can't say
6 it isn't, but it's just not strong evidence.

7 So trend is the key word here, not -- I
8 am not questioning that the high exposure category did
9 have this significant increase odds ratio. That's not
10 the intent here.

11 Q. I think I am not clear on what the
12 intent of your bullet Item 2 then was?

13 A. Well, we do a statistical test to
14 determine whether or not you find what is called a
15 trend in the data; that is, whether you see over a
16 range of exposures an increasing risk.

17 Now, the authors conclude that there was
18 a trend. I simply pointed out -- actually it was one
19 of my statisticians who pointed this out, it was really
20 sort of flat and then went up one data point to a high
21 odds ratio and that didn't seem to be strong evidence
22 of a trend.

23 Again, I don't make very much of this.
24 This is not a terribly important point. There was no
25 difference in the odds ratios for the first three

1 categories of exposures and then all a sudden you had
2 an increased odds ratio. That doesn't look like a
3 trend, even though statistically it satisfies certain
4 criteria for a trend.

5 Q. The trend is as reliable as the
6 statistics say it is; is that right?

7 A. I guess I don't understand the
8 question. You mean is a statistical test for trend the
9 only determinant of whether this is one?

10 Q. Let me go withdraw that question and
11 ask you a different one that might clear this up.
12 Would you agree with me that there was less than a five
13 per cent chance that the trend seen was only due to
14 random events?

15 A. By the statistical test, that is
16 correct.

17 Q. I wonder if you could also confirm
18 for me that in fact there was less than a .4/100ths
19 chance that the trend seen was only due to random
20 events?

21 A. May I look at the exhibit?

22 Q. Exhibit 754.

23 MR. CASSIDY: Do you have that, Dr.
24 Rodricks.

25 DR. RODRICKS: I do now.

1 MR. CASSIDY: Do you also have Exhibit
2 789?

3 MR. CASTRILLI: We are looking at page
4 1142, table -- sorry, I believe it's Table 1. In any
5 event, it's the only table on page 1142.

6 Q. Dr. Rodricks, I presume you are
7 looking at the final column on the right-hand side of
8 that table, the non-Hodgkin's lymphoma column?

9 DR. RODRICKS: A. Yes, I'm looking at
10 table -- the only table, as you said, I can't read it,
11 on page 1142. I can't read the number of the table.
12 And in the table under Non-Hodgkin's Lymphoma they have
13 categorized exposures among lymphomas according to the
14 frequency that they reported using herbicides in
15 general.

16 Q. Dr. Rodricks, when I look -- I'm
17 sorry, were you finished?

18 A. Yes, I can answer the question. They
19 have one, two, three, four five exposure categories if
20 you look at that listed in days per year, 0, 1 to 5, 6
21 to 10, 11 to 20 and greater than 21. If you look in
22 the far right-hand column you will see the odds ratios
23 they reported.

24 Now, for the first four --

25 Q. Dr. Rodricks, perhaps for the record

1 you can simply identify the four odds ratios you are
2 referring to so it is clear in the transcript?

3 A. Yes. I am looking under the last
4 column, Non-Hodgkin's Lymphoma, and for the frequency
5 of use category zero, the odds ratio was 1.3. Do you
6 see that?

7 Q. Yes. Please continue.

8 A. Those are farmers. You will notice
9 that the farmers themselves without any herbicide
10 exposure had an increase above the non-farmers.

11 Then 1 to 5 days a year, the odds ratio
12 was 1.4 and if you look at the confidence interval, not
13 different from the first; 6 to 10 days per year it was
14 12.6, again, not different from the first two; 11 to 20
15 days per year, 2 .6, again statistically not different
16 from the first three; then the one we were referring to
17 in the text, more than 21 days per year, it jumped to
18 six and that one was statistically different from the
19 1.3 and that is the association in this study that has
20 caused the concern.

21 Now, when you do a statistical test, a
22 so-called chi-square test for trend, it is
23 statistically significant at the .0004 level. That is
24 highly significant.

25 My statement in the excerpt -- my

1 statement here simply notes that even though that is
2 statistically significant as a trend and I'm not --
3 that's quite clear that it is, just note that the first
4 four exposures were basically flat and then you have a
5 jump in the odds ratio.

6 That doesn't look like a trend one, would
7 expect to see some increase in risk with increasing
8 exposure, but I'm certainly not denying that that is
9 statistically speaking a trend and certainly strongly
10 influenced by the odds ratio in the high exposure
11 group, not by the others.

12 Q. Dr. Rodricks, I am interested in your
13 use of the term flat. If we look at the five numbers
14 you just identified into the record, 1.3, 1.4, 1.6, 2.6
15 and 6.0, if we were to plot that on a graph would that
16 look like a flat line to you?

17 A. Well, just remember that the 1.3 and
18 the 2.6 are indistinguishable statistically by these
19 tests. Notice the lower confidence limit on both is
20 .8. Remember, the zero exposure had 94 cases, whereas
21 when we were dealing with the 11 to 12 days per year,
22 there are only five cases there. So the confident is
23 fairly wide.

24 If you were to plot them, they might look
25 like a gradual increase. They would look like a

1 gradual increase if you plotted them without the
2 confidence intervals, yes.

3 Q. So it is clearly a trend; isn't that
4 right?

5 A. I wouldn't call it a trend and I have
6 seen other real trends in the data, but I'm not going
7 to argue with you if you prefer to call it a trend.

8 Q. Well, it's your evidence we care
9 about, Dr. Rodricks, I am not in a position to give
10 evidence.

11 A. Well, when I've seen instances of a
12 real trend -- I mean, I've been recently through the
13 benzene epidemiology data and there you see, in this
14 -case, worker studies, rubble workers exposed to benzene
15 get leukemia, and there are since several studies one
16 can see increasing exposure. You get clearly increased
17 levels of risk.

18 That's a pretty strong trend in the data
19 or the response relationships. I have seen the same
20 thing with arsenic exposures by inhalation.
21 Statistically speaking this a trend. I am not going
22 to -- obviously not going to debate that.

23 Q. Thank you. I think we are still on
24 page 53 of your evidence. You are discussing on that
25 page several uncertainties and I just want to clear I

1 understand what it is that you are concerned about
2 here.

3 When I look at all six of the points you
4 identify between pages 53 to 57, taken as a whole, is
5 it your testimony that the Kansas study did not rule
6 out all the possible causes of non-Hodgkin's lymphoma?

7 A. Are you on page 54?

8 Q. Actually, I was taking --

9 A. The conclusions?

10 Q. I was really taking pages 53 through
11 57 together as a whole. Would a fair statement of your
12 position as expressed in those pages be that you are
13 saying the Kansas study did not rule out all other
14 possible causes of non-Hodgkin's lymphoma?

15 A. In this particular group, that's
16 correct.

17 Q. And is that what you mean when you
18 say at page 56 of your evidence -- I guess it really
19 summarizes it. At the top of the page:

20 "In short, no causal connection between
21 the use of 2,4-D and increased risk of
22 NHL was established."

23 A. That's my ultimate conclusion if you
24 were pointing to 2,4-D specifically, yes.

25 Q. Now, isn't it essentially impossible

1 for any single epidemiology study to meet that
2 standard?

3 A. Yes, I think I went through that.

4 MR. CASTRILLI: Madam Chair, do we break
5 at three o'clock?

6 MADAM CHAIR: 3:10, Mr. Castrilli?

7 MR. CASTRILLI: 3:10, I'm sorry. I don't
8 know why I fix on hours like that.

9 MR. CASSIDY: Just while we are waiting
10 for Mr. Castrilli, could the witnesses confirm that you
11 have Exhibit 789?

12 DR. RACHMAN: What is that, Mr. Cassidy?

13 MR. CASSIDY: The letter from...

14 DR. RACHMAN: I am sure I have seen that
15 here somewhere.

16 MR. CASSIDY: All right, thank you.

17 DR. RACHMAN: Yes. Dated August 28, 1985
18 to Ms. Kathleen Murphy.

19 MR. CASSIDY: Yes, thank you.

20 MADAM CHAIR: Dr. Rodricks, we've had
21 testimony from one witness who referred to the fact
22 that 2,4-D has been used going back to the early 1940s.
23 It has been the case with some chemical agents that one
24 problem in studying them is the fact that they haven't
25 been in use for that long and you have to wait for

1 cancers to develop because they haven't been in the
2 market or they haven't been used for a long period of
3 time.

4 How would you see the very long record of
5 use of 2,4-D fitting into the possibility that it is a
6 carcinogen; in other words, the fact that it has been
7 in use for 40 or 50 years, would you expect to be able
8 to find cancers more easily because of its widespread
9 use?

10 DR. RODRICKS: The general answer to that
11 is yes, for sure. It came into use I think in about
12 '47 and was used very widely, so you have 40 plus years
13 of use.

14 I guess -- I can't remember or recall
15 whether in all of these various studies where they
16 looked at populations what the earliest exposure might
17 have been in those populations. Some of them go back
18 into the 50's for sure.

19 So the latency here, I guess, in some
20 cases has been quite long. We're not sure in some of
21 the studies how long people might have been exposed.
22 That is a bit problematic.

23 MADAM CHAIR: Nor do we have details on
24 the formulations, but...

25 DR. RODRICKS: That's correct. The other

1 thing, I think what is difficult about all this is that
2 it wasn't just 2,4-D but other phenoxy herbicides that
3 came into use at the same time, particularly 2,4,5-T,
4 which is now no longer used and was probably a more
5 important material during the 50s and 60s and that
6 confounds interpretation of almost all of these
7 studies.

8 MR. CASTRILLI: Q. Page 58 of your
9 evidence. Dr. Rodricks, here you are reviewing the
10 results and your interpretation of the Western
11 Washington Study also known, I guess, as the Woods
12 study in other contexts.

13 As I understand your testimony on this
14 page, the Woods study failed to find an increase in the
15 risk of either soft tissues sarcoma or non-Hodgkin's
16 lymphoma for users of phenoxy herbicides. Is that your
17 testimony?

18 DR. RODRICKS: A. Or chlorophenols.
19 They looked at the two combined, chlorophenols are a--

20 Q. Right. For the purposes of --

21 A. --separate class of chemicals, but
22 they looked at the two combined.

23 Q. All right, thank you. And as I
24 understand a further conclusion you draw, which is
25 expressed at page 59 of your evidence, the Woods study

1 must be considered a negative study with respect to
2 finding an association between 2,4-D and either NHL or
3 STS. Is that still your testimony?

4 A. Yes.

5 Q. The Woods study did find increased
6 risks for those potentially exposed to
7 phenoxyherbicides in any occupation for 15 years or
8 more during the period prior to 15 years before cancer
9 diagnosis; is that correct?

10 A. That was the one increased risk they
11 found, yes, related to phenoxies in general. I would
12 just like to add, no information about 2,4-D
13 specifically at all.

14 Q. I believe that Mr. Cassidy actually
15 filed that study. You are, of course, familiar with
16 it; is that right, Dr. Rodricks?

17 A. I have read it, yes. I have studied
18 it.

19 MR. CASTRILLI: Madam Chair, I would like
20 to make this the next exhibit. It is entitled Soft
21 Tissue Sarcoma and Non-Hodgkin's Lymphoma in Relation
22 to Phenoxyherbicide and Chlorinated Phenol Exposure in
23 Western Washington, by James S. Woods et al. It
24 appeared in the journal of the National Cancer
25 Institute, Volume 78 in May 1987.

1 MADAM CHAIR: That's Exhibit 1247.

2 ---EXHIBIT NO. 1247: Document entitled Soft Tissue
3 Sarcoma and Non-Hodgkin's
4 Lymphoma in Relation to
5 Phenoxyherbicide and Chlorinated
Phenol Exposure in Western
Washington, by James S. Woods et
al.

6 MR. CASTRILLI: Madam Chair, I wonder if
7 this might be an appropriate place for a break to give
8 Dr. Rodricks an opportunity to scan it again. I know
9 he is familiar with it, but, in any event, it might
10 give him an opportunity to reconsider his position.

11 Would this be an appropriate place for a
12 break?

13 MADAM CHAIR: Yes, I think you want to
14 break, Mr. Castrilli. We will take one right now.

15 MR. CASTRILLI: All right, thank you.

16 MR. CASSIDY: Madam Chair, I wonder if I
17 could ask if Ms. Devaul could provide me with a copy of
18 Exhibit 1233.

19 Mr. Castrilli has indicated he intends to
20 cross-examine the witnesses on this and we have been
21 spent the last hour and a half trying to locate our
22 copy and we are having some difficulty and if we could
23 at least borrow an extra one of the Board's copy, the
24 witnesses will --

25 MADAM CHAIR: And that's 12...?

1 MR. CASSIDY: 33.

2 MR. CASTRILLI: Madam Chair, it is
3 another telephone directory size document known as the
4 Weeks report. Mr. Martel has it in his hand.

5 MR. CASSIDY: I have a feeling what
6 happened is our exhibit is with the BEAK witnesses who
7 were cross-examined at length.

8 MR. CASTRILLI: Does that mean it's in
9 Mexico?

10 MADAM CHAIR: Why don't you take this
11 copy.

12 MR. CASSIDY: Thank you very much, Madam
13 Chair.

14 Mr. CASTRILLIS: Thank you.

15 MADAM CHAIR: The Board will be back in
16 20 minutes.

17 ---Recess taken at 3:10 p.m.

18 ---On resuming at 3:35 p.m.

19 MADAM CHAIR: Please be seated.

20 MR. CASTRILLI: Madam Chair, if you will
21 give me one moment, I think my microphone melted over
22 the break. I want get it back up to the right
23 position.

24 Q. Dr. Rodricks, we were discussing
25 before the break -- or about to begin discussing before

1 the break the West Washington study.

2 DR. RODRICKS: A. Yes.

3 Q. And your commentary on it in your
4 evidence, which is at pages -- or is found at pages 58
5 and 59. And I have now introduced as an exhibit the
6 study that was under discussion, the Woods study and
7 that is now Exhibit 12...

8 MADAM CHAIR: 47.

9 MR. CASTRILLI: 1247.

10 Q. Just as a preliminary matter, can you
11 confirm for me that the -- I will call it the Woods
12 study just so you will understand what I mean by that.

13 I understand that the Woods study
14 specifically found that estimated risks of NHL were
15 elevated among forestry herbicide applicators. Is that
16 your understanding?

17 DR. RODRICKS: A. Well, that's one of
18 many, many findings in the study, not the only one.

19 Q. I wasn't suggesting it was the only
20 one, but it is you're understanding that that is one of
21 the findings of the study; is that correct?

22 A. Yes.

23 Q. And can you confirm --

24 A. You did say forestry--

25 Q. Yes, I did --

1 A. --because there are many other
2 spraying occupations with no elevated risk. Did you
3 say forestry or farmland?

4 Q. I said forestry.

5 A. Okay. Yes, with forestry they did
6 find high elevation.

7 MR. MARTEL: Can we repeat it, please?

8 MR. CASTRILLI: I'm sorry, Mr. Martel,
9 were you asking me to repeat the question so we are
10 sure about Dr. Rodricks' answer?

11 MR. MARTEL: Yes.

12 MR. CASTRILLI: Okay, I will do that.

13 Q. Dr. Rodricks, can you confirm for me
14 that the Woods study specifically found that estimated
15 risks of NHL, that's non-Hodgkin's lymphoma, were
16 elevated among forestry herbicide applicators?

17 DR. RODRICKS: A. That is correct.

18 Q. Thank you. And would you agree with
19 me, Dr. Rodricks, that spraying forests with
20 phenoxyherbicides gave the highest risk? Is that your
21 understanding?

22 A. Well, among the occupational risks
23 that they looked at, spraying forests with herbicide in
24 general gave the highest risk, yes.

25 Q. And we see that --

1 A. That's just among the occupational
2 risks.

3 Q. Yes, and that's the context in which
4 the question was asked. And we see that reflected, for
5 example, at page 899 in the abstract, in the middle of
6 the abstract. And more specifically, would you agree
7 with me, Dr. Rodricks, we see it reflected at page --
8 Table 4 at page 903?

9 A. Yes, Table 4 is where they summarize
10 their analysis of risks associated with various
11 occupations in which phenoxyherbicides are used and
12 they categorize the occupation or activity in that
13 table according to whether they believe it is
14 -relatively low, medium or more intense exposure to the
15 herbicides, then they list the odds ratio, as you can
16 see, for soft tissue sarcoma and NHL.

17 Q. All right. And if we look at the
18 heading for NHL under phenoxyherbicides and we go down
19 the column to spraying forests with herbicide, this is
20 under the high exposure occupation, the finding was an
21 odds ratio of 4.80? That would be 4.8 times the risk
22 of someone not exposed; is that your understanding of--

23 A. That's correct.

24 Q. --how to read Table 4?

25 A. That's correct.

1 Q. Thank you. Can I refer you in
2 Exhibit 1247, that's the Woods study -- I'm sorry, we
3 are still with the Woods study, to page 907.

4 MADAM CHAIR: One question, Dr. Rodricks.
5 What was the total study population?

6 DR. RODRICKS: This was called a case
7 control study where they selected cases of people with
8 non-Hodgkin's lymphoma, who had it or had died from it.
9 The total number of cases is listed here.

10 MADAM CHAIR: So is that the 128 with STS
11 and the 576 with --

12 DR. RODRICKS: Yes, they had 576 NHL
13 cases.

14 MADAM CHAIR: And the per cent study
15 population column refers to the number of forestry
16 workers being one per cent of--

17 DR. RODRICKS: Yes, the size of the
18 various worker--

19 MADAM CHAIR: --the 576?

20 DR. RODRICKS: --populations that you see
21 in Table 4, you could multiply that per cent by the
22 total number of cases to get the number of NHL cases
23 considered there.

24 MADAM CHAIR: So it's one per cent?

25 DR. RODRICKS: That's why the

1 confidence -- if you look at the confidence intervals
2 on the 4.8 figure they are fairly wide. I guess that's
3 probably six cases.

4 MADAM CHAIR: Okay, thank you.

5 MR. CASTRILLI: Q. Dr. Rodricks, we are
6 now turning our attention to page 907 of the Woods
7 study.

8 DR. RODRICKS: A. Yes.

9 Q. Under this is under the general
10 section of the report dealing -- well, it's the
11 authors' discussion of their findings or a portion of
12 their discussion with respect to their findings.

13 And would you agree with me, Dr.
14 Rodricks, that the Woods study indicates that it may be
15 hard to find a control population because dioxins and
16 furans are so widespread in American and Canadian
17 populations and that this may account for the
18 differences in risk estimates between the Woods study
19 and the Swedish studies?

20 A. I didn't get to this part of the
21 discussion. I don't recall it very well. I would
22 really need to read it again.

23 Q. Why don't we read it together.
24 Looking at column two on page 907?

25 A. Yes.

1 Q. The paragraph that begins:

2 "Differences..."

3 A. Yes.

4 Q. Do you see it?

5 A. Yes.

6 Q. Maybe I will just read it into the
7 record.

8 "Differences in risk estimates observed
9 between this and the Swedish studies
10 might also be accounted for on the basis
11 of variation in the extent of
12 non-occupational exposure received by the
13 general populations in the areas where
14 the studies were conducted. Several
15 investigators..." and these are referred
16 to in references 50 and 51,

17 "...have recently reported widespread
18 contamination of the general population
19 in the United States and Canada with
20 PCDDs...."

21 Just stopping there, Dr. Rodricks, that
22 would be dioxins?

23 A. Yes, that's the broad category of
24 polychlorinated dioxins. Yes.

25 Q. "...and PCDFs..." That would be the

1 broad category of furans?

2 A. That's correct.

3 Q. "...based on analysis of human fat
4 samples. The findings indicate that
5 although higher levels of total dioxins
6 and other contaminants may be seen in
7 some exposed persons, there is
8 considerable overlap in actual tissue
9 concentrations of such substances between
10 some person's with confirmed occupational
11 exposures and others who are not
12 previously known to have been exposed
13 through job related activities. These
14 observations suggest that epidemiologic
15 studies conducted in areas where the
16 extensive use of phenoxyherbicides and
17 chlorophenols has occurred may have
18 inadvertently included subjects who have
19 experienced significant exposure to the
20 chemicals of concern outside of the
21 occupational setting. Should this be the
22 case, it is possible that estimates of
23 actual risk based on recall of
24 occupational exposures alone may be
25 underestimated, owing to non-differential

1 misclassification of subjects according
2 to exposure status."

3 And there is a reference 52 at the end of
4 that paragraph.

5 A. Yes.

6 Q. In general, Dr. Rodricks, do you
7 agree with the statement I have just read into the
8 record?

9 A. Well, I don't -- I thought I knew
10 pretty well the data on background levels, so-called;
11 that is, levels of these materials and people not
12 occupationally exposed. There are in the five to ten
13 parts per trillion range in most of us.

14 The statement I am wondering is
15 'considerable overlap'. Let me see the exact words.

16 Q. Sorry, perhaps you could direct
17 our --

18 A. "...considerable overlap in actual
19 tissue concentrations of such subjects
20 between some persons with confirmed
21 occupational exposure and others not
22 occupationally exposed."

23 I would have to see the data they are
24 referring to there. That doesn't strike true to me.
25 Occupational exposures tend to be considerably higher,

1 but let me say, what they are trying to do here is to
2 explain the difference in observation, the striking
3 difference in observation here from Sweden.

4 I see most of this discussion as setting
5 forth some hypotheses about why such a difference might
6 exist. They are assuming the dioxins or furans might
7 be involved in this without any real evidence that in
8 fact they are. It is a perfectly reasonable
9 hypothesis, but it is nothing more than that.

10 If it's true, we would have to agree with
11 its conclusion that you could obscure a risk if your
12 background population indeed had in the U.S. and Canada
13 a higher background risk. That would be true. But
14 this is very conjectural and it is more hypothesis
15 generated.

16 Q. With that caveat, Dr. Rodricks, do
17 you agree with the paragraph?

18 A. If it is true that dioxins are
19 causally related to these conditions and if it is true
20 that the general level and background population does
21 overlap with the occupational background level, and I
22 am skeptical about that, but if that is true, that
23 would tend to reduce the sensitivities of these tests
24 in the U.S. or Canada to protect effects. That would
25 be true, but only if the first conditions are correct

1 and I am not sure I accept that.

2 Q. Would you agree with me, Dr.

3 Rodricks, that the assessment that I just read into the
4 record suggests possible problems with the Woods
5 studies overall conclusion that you rely upon?

6 In particular, let me continue so you
7 understand the context of the question. You state
8 that the Woods study did not find an increase in the
9 risk of either STS or NHL among users of
10 phenoxyherbicides.

11 Would you agree with me that the
12 assessment that I just read into the record at page 907
13 suggests possible problems with the Woods study overall
14 conclusion that you rely upon, in particular the one I
15 just suggested?

16 A. I think I would go so far as to say
17 that if this were correct, which you read in the
18 paragraph, it might explain why an excess might be
19 found in Sweden and not here. That does not
20 necessarily create -- that is not a problem in the
21 Wood's study, it is an attempt to explain the
22 difference in findings.

23 You are saying that the Woods study would
24 have less sensitivity to detect effect than would the
25 Swedish studies. It is not the fault of the study.

1 Q. Isn't the possibility raised in that
2 paragraph, Dr. Rodricks -- I apologize, I almost called
3 you Dr. Woods.

4 Isn't the possibility raised in this
5 paragraph, Dr. Rodricks, that the Woods studies
6 estimates of risk of cancer from phenoxyherbicides
7 including 2,4-D may have been underestimated?

8 A. If this is all true, I agree, yes,
9 relative to what was estimated in Sweden.

10 Q. And that's because of the confounding
11 factors described in the passage I just read to you; is
12 that right?

13 A. That's correct.

14 Q. Let's continue with page 907. We are
15 now looking at the last paragraph on the page. I am
16 not going to read the entire paragraph, it goes on for
17 some time on to page 907. I am just going read the
18 first portion of that paragraph.

19 "To estimate the..."

20 Sorry, do you have the paragraph? It is
21 the one that begins: "To estimate..."

22 A. Yes, I see it.

23 Q. "To estimate the extent to which
24 non-occupational exposure to
25 phenoxyherbicides may have occurred in

1 the present investigation, we have
2 evaluated data from several air
3 monitoring studies..." and the authors
4 refer to references 53 and 54 in this regard,
5 "...conducted during the spraying season
6 in the Pacific Northwest. These data
7 indicate that phenoxyacetic acids..."
8 Just stopping there there. Dr. Rodricks,
9 my understanding is that's another terminology for
10 phenoxyherbicides?

11 A. Yes.

12 Q. "...as well as PCDDs...

13 That's the general category of dioxins?

14 A. Yes.

15 Q. "...can transported in the atmosphere
16 either as vapour or adsorbed on particles
17 for distances ranging from several
18 hundred feet up to a mile from the
19 application area..."

20 There is a further reference, reference

21 54,

22 "...depending on weather conditions and
23 mode of dispersion."

24 Now, just stopping there, Dr. Rodricks,
25 is that an assessment you agree with?

1 A. The reference is a study I have not
2 read, reference 54.

3 Q. Do you have any better information?

4 A. No.

5 Q. And let's just hypothesize again, if
6 we might for a moment, Dr. Rodricks. Would you agree
7 with me that if this is true, then this would again
8 underscore the difficulty in finding a control
9 population because these chemicals are so widespread in
10 the general non-occupational human population?

11 A. If the assumption is that the dioxins
12 are what are responsible here for any observed
13 excesses, then this would be correct. What you say
14 would be correct.

15 MADAM CHAIR: Excuse me, Dr. Rodricks.
16 Why wouldn't the assumption work equally well in the
17 other direction, that there would be -- that a control
18 would in fact control for everyone, for all the
19 populations exposure to any dioxins?

20 DR. RODRICKS: Well, I guess if I think
21 it's true, the assumption here is that if individuals
22 have collected in their body dioxins, which remain in
23 the body, but the phenoxyacetic acids do not, they are
24 rather quickly excreted, but if dioxins are accumulated
25 in the body there would be an exposure there.

1 It is not zero in that population, but if
2 it increased and there's potentially higher background
3 risk, if you like, in the so-called controls, if the
4 risk is really due to these dioxins such that -- that
5 would tend to obscure a difference between controls,
6 because they are not zero controls, they have some
7 finite body burden as against people who have clearer
8 and obvious occupational exposures.

9 MR. CASTRILLI: Q. Dr. Rodricks,
10 continuing with you. Would you agree with me that risk
11 estimates like the ones in the Woods study, based only
12 on assessment of occupational exposures without asking
13 about residential or home use exposures, could be
14 underestimated as a result of exposure in this
15 classification.

16 DR. RODRICKS: A. Home use exposures,
17 yes, that could be a confounding factor in the control
18 rooms.

19 Q. If I could just refer you to page 908
20 in this regard. We are looking at a slightly -- sorry,
21 we are looking at what would be the left-hand column on
22 the page, slightly more than halfway down the sentence
23 beginning: "Nevertheless... "

24 Do you see that?

25 A. No. 908?

1 Q. Sorry, page 908, left-hand column,
2 top left-hand portion of the page, halfway down that
3 paragraph begins the phrase:

4 "Nevertheless, should phenoxyherbicides
5 and/or their contaminants..."

6 Do you see that?

7 A. I'm sorry.

8 Q. Let me just point it out to you.

9 A. Yes, okay.

10 Q. The Woods study authors indicate:

11 "Nevertheless, should phenoxyherbicides
12 and/or their contaminants increase the
13 risk of cancer at environmental exposure
14 levels or, as recently suggested, produce
15 subclinical immune system alterations
16 that may predispose to such risks, it is
17 possible that risk estimates based solely
18 on assessment of occupational exposures
19 could be attenuated as a result of
20 exposure misclassification."

21 Do you agree with that assessment?

22 A. If all the premises are true, I don't
23 agree that all the premises are true. Their
24 hypothesizing what would result if, for example, there
25 were an accumulation of contaminants, such as dioxins,

1 in people who are not occupationally exposed and it
2 could alter immune systems, if that is true, and I
3 don't believe there is convincing evidence to support
4 that, but if that were true then there could be an
5 attenuation of risk -- of the risk estimates, assuming
6 that only the occupational exposures contribute to
7 disease. So the conclusions would be true if all the
8 premises are correct, but I don't agree with all the
9 premises.

10 I also have to note here, and it is
11 something I didn't remember on my first reading on your
12 last question, that they did test this hypothesis about
13 home use of phenoxyherbicides. I just noticed this, if
14 I may just go back to the previous--

15 Q. Please do.

16 A. --two sentences. They eliminated
17 subjects or they redid an analysis apparently who
18 reported home use exposures to phenoxyherbicides and
19 chlorophenols in the present study and that had no
20 effect on the estimated risks for either STS or NHL to
21 paraphrase that sentence. Do you see the sentence?

22 Q. Yes.

23 A. Hence, it is unlikely that bias due
24 to such exposures could account for the large
25 differences in risk estimates observed between these

1 and the Swedish studies. I had not recalled that they
2 had tried to do that, but that pertains to your last
3 question -- the question before last.

4 Q. Now, at page 59 of your evidence.

5 A. I'm sorry, may I -- I'm sorry, please
6 go ahead.

7 Q. At page 59 of your evidence you are
8 summarizing your position with respect to the Woods
9 study and you state, this would be the second paragraph
10 on the page:

11 "However, because the direct analysis of
12 phenoxyherbicides did not show an
13 association between this exposure and
14 disease, this must be considered a
15 negative study with respect to finding an
16 association between 2,4-D and either NHL
17 or STS."

18 I gather that's still your position; is
19 that right?

20 A. Yes, and that is based on all of the
21 data in this paper.

22 Q. Can I refer you in the Woods study to
23 page 901.

24 A. Yes.

25 Q. We are looking at the second full

1 paragraph.

2 A. "In coding occupational exposure...."

3 Q. Yes. The paragraph begins with that
4 phrase.

5 A. Yes.

6 Q. Firstly, can you confirm for me, Dr.
7 Rodricks, that the relative risks in the Woods study
8 are determined on a theoretical estimation of whether a
9 certain occupation is associated with low, medium or
10 high exposure?

11 A. Excuse me one moment. Now, what is
12 your question? Theoretically...

13 Q. Sorry, let me repeat the question.
14 Can you confirm for me that the relative risks in the
15 Woods study are determined on a theoretical estimation
16 of whether a certain occupation is associated with low,
17 medium or high exposure?

18 A. Well, they collected information for
19 each person on the job held and the use of
20 phenoxyherbicides and chlorophenols. They made an
21 assumption that there couldn't have been exposure prior
22 to the date when they were first marketed. They say
23 that in this paragraph, and then they say:

24 "The coding of each job episode held by a
25 study subject according to intensity

1 and duration of exposure..."

2 That was the coding they did when they
3 collected the information,

4 "...permitted evaluation of the exposure
5 history of each subject in terms of
6 duration of continuous or cumulative
7 exposure at each dose level. Thus a
8 complete exposure profile on each subject
9 for each class of chemical under
10 evaluation was obtained."

11 That's all they say on that topic. I
12 take their word that that's what they did.

13 Q. I think it might have been helpful if
14 I had directed you to another passage in the paper.
15 Let me do that now.

16 A. All right.

17 Q. Page 900.

18 A. All right.

19 Q. We are looking at the right-hand
20 column near the bottom. We see the heading -- sorry,
21 we see the sentence that begins: "Each job title..."

22 Do you see that?

23 A. Yes.

24 Q. I will just read that into the
25 record.

1 "Each job title or activity was then
2 assigned to a 'high', 'medium', 'low' or
3 'no' exposure category for both
4 phenoxyherbicides and chlorophenols,
5 reflecting the consensus of the
6 consultant group of the likely intensity
7 of exposure to each chemical received in
8 that occupation. Examples of
9 occupational activities in each
10 exposure category are given in the
11 tables."

12 A. That is correct. That is quite
13 typical of these kinds of retrospective studies.
14 That's quite typical.

15 Q. Now, let's just look at the paragraph
16 I referred you to earlier which is the second full
17 paragraph on page 901, the paragraph beginning: "In
18 coding occupational exposure..."

19 Do you have that?

20 A. Yes.

21 Q. "In coding occupational exposure
22 to phenoxyherbicides..." It says:
23 "...based on job descriptions..."

24 Is that your understanding, the coding
25 was done on the basis of job descriptions?

1 A. Yes, and you see those in Table 4.
2 That is the actual job descriptions.

3 Q. Let's look at Table 3 first, which is
4 on page 902 at the bottom of the page.

5 A. Yes.

6 Q. If we just look, Dr. Rodricks, at the
7 italics associated with that table -- let me just read
8 that into the record.

9 " Risk (pooled odds ratio) of developing
10 STS or NHL in men...exposed...20-79..."
11 that's 20 years to 79 years,

12 "...by estimated intensity of past
13 occupational exposure to
14 phenoxyherbicides..."

15 Just stopping there, Dr. Rodricks. Can
16 you confirm for me that the relative risks in the Woods
17 study are determined on a theoretical estimation of
18 whether a certain occupation is associated with low,,
19 medium or high exposure and not on whether the
20 interviewee reported personal low, medium or high
21 exposure?

22 A. Right, the interviewee would have no
23 way to know that, no one has any way to know that and
24 that's why they turned to consultants who understand
25 these jobs and the activities and, again, this is

1 fairly typical in occupational studies where you are
2 trying to reconstruct historical exposures. There is
3 nothing much else you can do.

4 Q. So would it be fair to say, Dr.
5 Rodricks, that what the Woods study did was determine
6 exposure by job title and not by interview?

7 A. Well, they determined occupational
8 history by interview job categories and then they
9 ranked -- and also asked questions about the use of
10 phenoxyherbicides and other chemicals, chlorophenols,
11 and then ranked them using consultant experts who know
12 about these occupational jobs categories and the
13 relative intensities of exposure.

14 Q. Well, just let me be clear on your
15 response to my question. What the Woods study did was
16 determine exposure by job title; is that right?

17 A. Yes, and that's true of almost every
18 study there is on these herbicides.

19 Q. And would it be fair to say, Dr.
20 Rodricks, that the Woods study consultants assigned
21 people to particular exposure categories by occupation
22 and not by reported or demonstrated exposure?

23 A. Well --

24 Q. I'm sorry, let me finish the question
25 so you understand the context. In fact, let me repeat

1 the question so you understand the context.

2 Would you agree with me that Woods
3 assigned people to particular exposure categories by
4 occupation and not by reported or demonstrated exposure
5 and he pooled all of these folks together in terms of
6 their exposures?

7 A. We could just go back to what we have
8 already read into the record. They used a
9 questionnaire to identify occupations and activities
10 that involved the use of phenoxyherbicides and/or
11 chlorophenols. They used consultants to identify those
12 categories of jobs and they identified, as it says here
13 in the paragraph you referred to on page 900, 34
14 specific job titles, 17 job activities that involved
15 potential exposure to phenoxyherbicides or
16 chlorophenols, and then they categorized those
17 according to intensity of exposure that would be
18 associated with those kinds of jobs or activities.

19 So, yes, I think we have said this
20 already. That's what they did.

21 Q. I want the record to be clear on
22 this. Let me restate it a slightly different way and
23 see if your answer still is the same.

24 Woods pooled exposures together for all
25 those with only one time exposure or a few exposures to

1 many exposures and he put those within a category; is
2 that right?

3 A. Would you repeat that?

4 Q. I'm sorry?

5 A. Say that again.

6 Q. Yes. Woods pooled exposures together
7 for all those with only one time exposure, a few
8 exposures to many exposures and put those within a
9 category; in other words, he might have had -- if you
10 can graphically see this, he might have put a range of
11 exposures within the low category, he might have put a
12 range of exposures within the medium category and he
13 put a range of exposures in the high category and we
14 don't know what those are; is that right?

15 A. No, he doesn't say anymore than we
16 relied upon the work of the consultants to do that.

17 Q. Would you agree with me, Dr.
18 Rodricks, that serious dilution would occur by lumping
19 cases together on the basis of their exposure -- excuse
20 me, on the basis of their occupation rather than on
21 their reported recalled exposure? Is that a
22 possibility?

23 A. It's possible if there are serious
24 areas made in the exposure categorization, yes.

25 Q. Let's continue with page 901. This

1 is the first full paragraph on page 901. As I
2 understand what the Woods consultants did from a
3 reading of the study, is that they asked the
4 interviewees about their extent of exposure to specific
5 chemicals of interest and the precise time intervals
6 during which each exposure episode had occurred.

7 That's your understanding as well; is
8 that right?

9 A. That's what they attempted to do,
10 yes.

11 Q. Having done that, would you agree
12 with me, Dr. Rodricks, that nowhere in Woods study are
13 any relative risks calculated on the basis of these
14 responses, whatever they were, because Woods does not
15 in fact report on what they were?

16 A. Can you give me a few moments here?

17 Q. Sure, please take your time.

18 A. My assumption in all of this analysis
19 was that -- the three paragraphs we referred to
20 describe the total activity they went through to
21 categorize workers' exposure based on job categories
22 and interview in which people described their jobs, job
23 titles and other information they could collect, and
24 then they assigned people according to that collective
25 informing, and that the analyses, insofar as it deals

1 with exposure versus risk, included that total
2 analysis. That's been my interpretation all along
3 here.

4 Q. Do you want another moment to review
5 the document?

6 A. Your question is whether the
7 categories in Table 3, low, medium and high were based
8 strictly on information supplied by the consultants?

9 Q. I wouldn't --

10 A. Is that your question?

11 Q. I wouldn't put it that way. Let me
12 rephrase the question I asked earlier and see if we can
13 shorten this up.

14 A. Please.

15 Q. The proposition I was putting to you,
16 Dr. Rodricks, was that nowhere in the Woods study are
17 any relative risks calculated on the basis of these
18 responses, the responses we referred to at page 901,
19 whatever they were, because Woods does no report on
20 what these were; isn't that your understanding?

21 A. My understanding is that that's what
22 the categories in Table 3 refer to.

23 Q. The relative risks?

24 A. The odds ratios, mm-hmm.

25 Q. Where are the relative risks referred

1 to?

2 A. Well, under NHL, for example, the
3 exposure category. I am referring to Table 3 on page
4 902. The odds ratio is another name for relative risk.

5 Q. Sorry, I think we are two ships in
6 the night here and it probably my fault in terms of the
7 question. Where in the Woods study do the authors
8 calculate relative risks on the basis of the responses
9 they received?

10 A. Well, they do several different kinds
11 of analyses based on those responses. This is one.

12 Q. From the interviewees. Where do we
13 have the information from the interviewees in terms of
14 relative risks?

15 A. In Table 3 they constructed an
16 exposure category based on information about job title,
17 length of time in the job and information provided by
18 the interview and they state they were interviewed
19 1983-85, and then in Table 4 they have odd ratios for
20 the same group of men, but they break them out a
21 different way according to specific occupations or
22 activities and those occupations or activities are
23 broken into low, medium and high exposure categories.

24 Q. Dr. Rodricks, what I am going to do
25 is I am going to red flag that question and come back

1 to it because I think if we move a little further into
2 the Woods study it will become more apparent and then
3 we can come back to it.

4 A. We have some miscommunication here, I
5 don't understand your question.

6 Q. All right. Looking at page 901, this
7 is the second full paragraph in the middle of the page.

8 A. Yes.

9 Q. We are looking about halfway down in
10 that paragraph, the sentence that begins: "The coding
11 of each job episode..."

12 Do you see that? The word 'the' begins
13 on the right-hand -- sorry, the left-hand column, the
14 last word in the sentence. Let me assist you, if I
15 might.

16 A. "The coding of each job episode...."

17 Q. Yes, that's right. Let me just read
18 that into the record and we will pursue this.

19 "The coding of each job episode held by a
20 study subject according to intensity and
21 duration of exposure permitted evaluation
22 of the exposure history of each subject
23 in terms of duration of continuous or
24 cumulative exposure at each dose level."

25 Now, would you agree with me, Dr.

1 Rodricks, that if Woods in that paragraph I just read
2 into the record is referring to reported exposure this
3 is never in fact evaluated statistically in the Woods
4 study?

5 A. I assume that's what Table 3 is. Is
6 there some reason why -- point out to me why that would
7 not be the case.

8 Q. Because I think from the body of the
9 study it is clear that he was reporting exposure. As
10 he says in Table 3, for example, estimated on the basis
11 of job categorization, not on the basis of actual
12 exposure testing or assessment -- not testing,
13 assessment.

14 A. Well, it says this was based on
15 interviews and I don't know why you would collect all
16 that information in interviews and go to all that
17 trouble and then not use it.

18 I have been under the assumption the
19 exposure category, as listed in the Table B, would
20 represent that full analysis of job title and
21 information about exposures reported during --
22 collected the interviews. It says these were cases and
23 controls interviewed in the '83 to '85 period. So my
24 assumption is that's what they did.

25 Q. Let me just --

1 A. If that's wrong, I guess I don't know
2 why it would be wrong.

3 Q. Let me just ask you, then, your
4 understanding of where Woods is reporting reported --
5 sorry, your understanding of where Woods statistically
6 evaluates reported exposure is to be found in which
7 table?

8 A. Table 3 is one evaluation, Table 4 is
9 another evaluation and then there are several other
10 evaluations of odds ratios in the text for different --
11 they looked at many different kinds of exposures and
12 occupations.

13 Q. Let me put this proposition to you,
14 Dr. Rodricks. If Woods is talking about theoretical
15 exposure classification assigned to each occupation,
16 such a breakdown for each subject does not appear in
17 the statistic calculations.

18 I am thinking, for example, if you look
19 at Table 4 on page 903, we don't have a category such
20 as spraying woodlands with herbicides high exposure
21 versus spraying woodlands with herbicides brief or low
22 exposure. Would you agree with me we don't have that
23 kind of breakdown in Table 4?

24 A. Within some of the job categories?

25 Q. Yes.

1 A. They were unable to determine that.

2 Q. Your answer is...?

3 A. They were unable to determine that,
4 presumably.

5 Q. Thank you.

6 MADAM CHAIR: Excuse me, Mr. Castrilli, I
7 don't want to interrupt your cross-examination, but on
8 page 901 they do talk about some work that they did
9 with recall bias in terms of exploring specifically
10 with the interviewees where they couldn't be clear
11 about their exposure profiles and so they specifically
12 asked them for more information about it, specifically
13 for recall bias, but it must have helped refine a bit
14 what the specific kinds of exposures were in the
15 categories.

16 DR. RODRICKS: Yes.

17 MR. CASTRILLI: Madam Chair, what I am
18 exploring with this witness is whether in fact Woods
19 every reports on that in the body of the study, or
20 whether all he reports is the categorizations based on
21 the job title descriptions the consultants designed.

22 DR. RODRICKS: Well, my understanding is
23 that the exposure categories as set forth in Table 3
24 and otherwise described in the text and in Table 4, a
25 different way to look at them, was based on the

1 information collected from consultants and people who
2 were interviewed, combined information.

3 MR. CASTRILLI: Q. Can I refer you to
4 page 903 of the Woods report.

5 DR. RODRICKS: A. Yes.

6 Q. Can you conform for me, Dr. Rodricks,
7 that Woods calculated relative risks associated with
8 exposure specifically to 2,4-D and 2,4,5,-T and to
9 phenoxyherbicides in general as he indicates on the
10 left-hand column?

11 A. He reports somehow breaking out
12 information on 2,4-D and 2,4,5-T and phenoxies in
13 general and he calculates risks of .73, that is less
14 than one for 2,4-D, .98 for 2,4,5-T and .87 for
15 phenoxyherbicides in general.

16 What is not all clear is how he broke out
17 2,4-D. You can't tell from this paper how he was able
18 to separate the two. He does say that there is no real
19 basis for it given in the paper that I could see.

20 Q. And would you agree with me that he
21 apparently pooled everything from one time exposure to
22 heavy exposure for the calculations?

23 A. The overall calculation? The .87
24 figure for phenoxyherbicide includes the entire study
25 population, yes.

1 Q. And what we have in Table 4 on page
2 903 are estimates or potential exposure and not
3 necessarily actual exposure; is that right?

4 A. Yes, we have in none of the studies,
5 positive or negative, any estimate of actual exposure.

6 Q. I just want to be clear about one
7 last point with respect to this study.

8 The Woods study does not determine
9 relative risk on the basis of recalled high, medium or
10 low exposure; is that your understanding?

11 A. My understanding is that Table 3, as
12 I read it, includes all of the exposure information
13 they collected for interviews and for job
14 categorization.

15 Q. And Table 3 talks about estimated
16 exposure?

17 A. Surely they are estimated.

18 Q. Do you know whether Woods gathered
19 data on the frequency of use within a particular
20 occupation?

21 A. Well, that he does not report and I
22 assume he did not do that or he would have reported it.

23 In the occupation categories; that is,
24 Table 4, he just had broad categories and groups them
25 himself. Maybe that is what you were getting at. He

1 does using consultants group the job categories, if
2 that's what you mean, by high, medium or low exposure,
3 but there is no breakdown within those specific job
4 categories, that is correct.

5 I have assumed, and I don't see any
6 reason why it is wrong, that Table 3 would reflect
7 categorization by total intensity of exposure.

8 Q. Dr. Rodricks, with all of the
9 questions about the Woods study we have been discussing
10 for the the last few minutes, I would like to have your
11 very clear evidence on the record as to why you believe
12 this is a negative study?

13 A. Well, I don't believe it shows an
14 excess of NHL or STS related to exposure to 2,4-D. The
15 total evidence in the study does not show an excess.
16 I am not by any means saying this proves that these are
17 not carcinogens, not by any means. I am just saying it
18 does not show an excess.

19 The data in Table 3 where exposures are
20 categorized for phenoxyherbicides in general, the odds
21 ratios are listed there for STS and NHL and by their
22 categorization there is no significant increase in the
23 three exposure groups over the uncontrols, any of
24 those.

25 There are other -- we went over the data

1 on the entire population where the odds ratio was .87.
2 According to occupation; that is, if you look at Table
3 4, there is one occupation in which we have -- I'm
4 sorry, two occupations in which you have a
5 statistically significant excess: the farmer
6 categorized as medium exposure where the odds ratio was
7 1.33, was barely significant, spraying forest with
8 herbicides was clearly significant although a
9 relatively small number of cases, but nevertheless
10 significant, and all the others -- in none of the
11 others is there any excess at all.

12 As a matter of fact, the strongest excess
13 for NHL occurred in Table 6, even these are -- you have
14 be be very careful here too. A small part of the
15 population, but the strongest excess you would find in
16 Table 6 are those individuals who have some compromised
17 immune system. People who have been taking
18 immunosuppressant drugs have a relative risk of 10.9.

19 They looked at these other factors, too,
20 and I certainly wouldn't conclude that they prove that
21 immunosuppressant drugs cause NHL either, not by any
22 means. There are some elevated NHLs associated with
23 chemicals, chlordane and DDT in combination. That's in
24 Table 7, welding metal fumes, those kinds of industrial
25 exposures. Those are a few excesses, but altogether I

1 find this unconvincing with respect to -- certainly
2 with respect to 2,4-D and even with the
3 phenoxyherbicides altogether.

4 I just emphasize that. I don't want mean
5 to prove that they are not carcinogens, that's not my
6 implication in my statement, but this does not add
7 anything to the evidence.

8 Q. With all of the confounding factors
9 associated with this particular study, why isn't it
10 simply inconclusive as opposed to negative?

11 A. Negative here means no excess shown,
12 no significant association shown altogether and maybe
13 that would be a better way to say it. Negative may
14 be -- if by negative you are reading that this shows it
15 not to be a carcinogen, then negative is wrong. I
16 didn't mean it that way.

17 Q. I am wondering, Dr. Rodricks, if we
18 might refer -- I haven't talked about your evidence for
19 a while, I'd almost forgotten the principal document we
20 are here to focus on.

21 I wonder if I could direct your attention
22 to your exhibit again, it is Exhibit 1239, and we are
23 looking at the -- beginning at page 60.

24 A. What number is this?

25 Q. I'm sorry, your witness statement is

1 Exhibit 1239.

2 A. Oh.

3 Q. Some of these numbers become
4 significant for some of us during the course of the
5 hearing and we forget that they mean absolutely nothing
6 to witnesses who come and go.

7 We are looking initially Dr. Rodricks, at
8 page 60.

9 A. Yes.

10 Q. And we are looking at the last full
11 paragraph on that page.

12 A. Yes.

13 Q. You begin -- well, I will begin the
14 sentence in the middle of -- sorry, let me start by
15 comment again.

16 Let me just read the entire sentence into
17 the record so it is clear what we are talking about:

18 "...if the Swedish studies are not
19 included in the plot of probability
20 distributions of the ORs from the case
21 control studies, then the remaining
22 studies are found to cluster close to
23 unity, indicating no difference between
24 cases and controls in phenoxyherbicide
25 use with respect to STS or NHL."

1 And just stopping there, Dr. Rodricks.
2 You refer in this regard to Figure 1, which is found at
3 page 612, and then let me just continue with the
4 remainder of the comment I want your views on.

5 "Figure 1 illustrates the relative size
6 of the ORs from the five NHL studies
7 reviewed by Bond..."

8 And, Madam Chair, Bond is of course
9 Exhibit 715 in these proceedings.

10 "The comparison is somewhat crude in
11 that the definition of exposure was not
12 uniform for these studies. In addition,
13 the Hardell study combined NHL cases with
14 HD..."

15 HD is Hodgkin's disease; is that right?

16 A. Yes.

17 Q. "...so at the least, the confidence
18 limits would be wider if the report
19 focused upon NHL. Figure 1 demonstrates
20 that a causal relationship between
21 phenoxyherbicide use and cancer use has
22 not been established. Three of these
23 studies (Woods et al. 1987..."

24 That's now exhibit 1247,

25 "...Cantor et al. 1986, and Pearce et al.

1 1986) are negative (ORs cluster about
2 1.0). Of the two positive studies, the
3 methodology of the Hardell et al. (1981)
4 study is open to question, while the
5 results of Hoar et al..." also known as
6 the Kansas study, "...are not specific for
7 phenoxyherbicides."

8 Now, I would just like to focus on the --
9 initially on the first sentence of the last paragraph
10 on page 60 of your evidence where you say:

11 "...if the Swedish studies are not
12 included in the plot of probability
13 distributions of the ORs from the case
14 control studies, then the remaining
15 studies are found to cluster close to
16 unity, indicating no difference between
17 cases and controls in phenoxyherbicide
18 use with respect to STS or NHL."

19 Now, jsut stopping there. Unity equals
20 1.0; is that my understanding?

21 A. Yes.

22 Q. Now, let's look at Figure 61 --
23 excuse me, Figure 1 on page 61 of your evidence. Now,
24 Dr. Rodricks, if we do what you suggest on page 60 and
25 we eliminate --

1 A. Excuse me, this is what Dr. Bond did
2 in his paper.

3 Q. Am I right --

4 A. I am describing the Bond paper.

5 Q. Do you adopt his evidence -- or do
6 you adopt his methodology?

7 A. I may have some questions about what
8 he did, it's crude in some respects, but it's useful in
9 others and I think that's what we describe here.

10 Q. All right. Let's just focus on
11 Figure 1 and if you want make any qualifications that
12 you think are appropriate you can -- please do that.

13 Now, would you agree with me that if we
14 were to eliminate the Hardell studies from Figure 1
15 that that would still leave four ranges that cluster
16 close to 1.0, but even the average -- sorry, that
17 cluster closer to 1.0, but even the average of them is
18 still greater than 1.0?

19 A. Well, we must include the confidence
20 limits and perhaps a clearer demonstration is in the
21 Bond paper itself. Can we refer to that?

22 Q. We are going to.

23 A. Okay. The confidence limits for the
24 combined four that remain all go in the low end below
25 one. You combine them. That's clearer perhaps in the

1 Bond paper itself.

2 Q. Well, would you agree with me that
3 even if we eliminate the Hardell studies from Figure 1
4 every study listed on Figure 1 is elevated above 1.0?

5 A. Not statistically. Three are not and
6 one is.

7 Q. With respect on the odds ratio, Dr.
8 Rodricks?

9 A. Well, you have to look at the
10 confidence interval, that's what the bar represents.

11 Q. Well, let's just look at the odds
12 ratio for a moment. Would you agree with me that with
13 respect to the odds ratio every other study -- sorry,
14 and leaving Hardell aside, every study on that page is
15 elevated above 1.0?

16 A. That's correct.

17 Q. Thank you.

18 Q. Let's look at the Bond paper.

19 MADAM CHAIR: What is the exhibit number
20 of the bond paper, Mr. Castrilli?

21 MR. CASTRILLI: Actually, it is Exhibit
22 715 and I am looking for my copy of it.

23 Q. Dr. Rodricks, it might be easier if
24 we began at page 174 of Exhibit 715.

25 DR. RODRICKS: A. Yes.

1 Q. This is a table that Bond has put
2 together dealing with a number of the studies that you
3 have included in Figure 1. In referring to the Kansas
4 study, Dr. Rodricks, Bond indicates that the odds ratio
5 for the 170 NHL population based cases was 2.2 with a
6 95 per cent confidence limit of between 1.2 and 4.1.

7 Is that how you read Table 1 with respect
8 to the Kansas study?

9 A. Yes, sir, that's for the entire
10 population study.

11 Q. Actually, I'm sorry, what I should
12 have done is asked you to get your copy of Exhibit 754
13 at the same time. That's the Kansas study. I don't
14 know if your copies have numbers on them.

15 Just looking at the Kansas study, Dr.
16 Rodricks, this is Exhibit 754 --

17 MR. CASSIDY: Do you have that, Dr.
18 Rodricks?

19 DR. RODRICKS: Yes, I have it.

20 MR. CASTRILLI: Q. Could you help me
21 find where the Kansas authors report an odds ratio of
22 2.2.

23 MR. MARTEL: What table are we looking
24 at, Mr. Castrilli?

25 MR. CASTRILLI: Mr. Martel, we are

1 looking at Exhibit 754, the Kansas study, and I have
2 not referred the witness to a particular page. I would
3 like him to help me.

4 DR. RODRICKS: I can refer you in Exhibit
5 754 to page 1143, the table there. Bond derived that
6 figure from the table. I can't read the number of the
7 table.

8 MADAM CHAIR: Table 2.

9 DR. RODRICKS: Is it Table 2?

10 "Every used phenoxyacetic acids..."

11 So it is the overall figure for the
12 study. 2.2 odds ratio with a confidence interval of
13 1.2 to 4.1, that's what they use.

14 MR. CASTRILLI: Q. Now, doesn't Bond
15 assume for the purposes of their analysis that the
16 results of the five studies represent the same
17 underlying population at risk?

18 DR. RODRICKS: A. Well, they make the
19 assumption, but they also qualify that in their text
20 and they recognize that there is a certain crudeness in
21 this analysis. They probably do not result the same
22 underlying distribution.

23 Q. But isn't that assumption important
24 for Bond in order to permit him to average the case
25 control studies?

1 A. Yes, they average the studies by
2 giving them equal weigh and that assumes they come from
3 the same -- as the statisticians call it, underlying
4 distribution of diseases, and we don't know whether
5 that's true, that is correct. They state that as an
6 assumption in their analysis.

7 Q. There is no basis for that
8 assumption, is there?

9 A. Well, if in fact -- I don't know
10 whether there is or not. If in fact these excessess
11 all represent the very same biological phenomenon, then
12 there could be, but we don't know whether that's true.

13 Q. The studies that Bond refers to, at
14 least in theory, looked at populations exposed to
15 different levels of risk, isn't that right, in terms of
16 duration and frequency?

17 A. Different levels of...

18 If you mean there are diverse populations
19 in terms of their exposure patterns and risk patterns,
20 yes, they are.

21 What they were attempting to do was do
22 something a little more than just kind of a qualitative
23 weight of evidence study, we have used that term
24 before. In looking at all of the data combined, they
25 tried to do something a little bit more qualitative

1 here with the odds ratios in the various studies.

2 It is crude, as we say, on page 62 of our
3 report, but it was an attempt to do something here,
4 somewhat more systematic than simply looking at all the
5 data and making a qualitative judgment. I hope in our
6 report we qualified our confidence in this analysis.

7 Q. Yes, I think you indicated twice it
8 was crude and once it was misleading.

9 A. Well, it could be. The important
10 thing about it, I think, is what it seems to show,
11 crude as it is, is that you have basically one study
12 with this extraordinarily high risk report in Sweden
13 which seems to not match the total body of evidence
14 very well.

15 You have not seen that same striking
16 phenomenon in other studies. I mean, that's -- maybe
17 what they have done is kind of a roundabout way of
18 getting to that conclusion which might be obvious just
19 upon inspection, but they tried to do it a little bit
20 more systematically than others have.

21 Q. We've had a discussion this afternoon
22 about the Woods study which in your evidence you
23 describe as negative and we've talked about the
24 possible confounding factors in the Woods study, so
25 isn't there some difficulty in Bond doing what he did

1 in relation to the Wood study in suggesting there is no
2 basis for a relationship between what was found in
3 Sweden and what might have been found in western
4 Washington?

5 A. Well, keep in mind that one basis for
6 the difference between Sweden and Western Washington is
7 that 2,4-D of the phenoxyherbicides or the dioxins do
8 not cause cancer. That's one possible explanation for
9 the observation.

10 The authors, Woods and others were
11 looking for other possible explanations for the
12 difference and we don't know why the difference exists.
13 All of these studies that Bond looks at have
14 methodological limitations. I hope we made clear by
15 now that every epidemiology study does, and they're
16 different from one study to the other.

17 Q. Now, in your evidence you discuss the
18 Canadian farm operator mortality study. Actually, I
19 guess it is not called that anymore. What is the
20 title?

21 MR. CASSIDY: The title is rather
22 lengthy, Madam Chair.

23 MADAM CHAIR: Did we make this an exhibit
24 yet, Mr. Cassidy?

25 MR. CASSIDY: Yes, it is Exhibit 1244.

1 MADAM CHAIR: 1244. Thank you.

2 MR. CASSIDY: Do you intend to break at
3 five o'clock tonight, Madam Chair?

4 MADAM CHAIR: Yes, Mr. Cassidy.

5 MR. CASSIDY: Thank you.

6 MR. CASTRILLI: Q. Dr. Rodricks, we
7 have -- sorry, in looking at the Wigle study, being
8 Exhibit 1244, would you agree with me that in this --
9 this is a cohort study, as I understand it, of male
10 Saskatchewan farmers, significant dose response
11 relationships were noted between risk of non-Hodgkin's
12 lymphoma and acres sprayed with herbicides.

13 DR. RODRICKS: A. Yes. You are reading
14 from the abstract?

15 Q. Yes. Well, actually it is found in
16 several places in the report, that's pme place it's
17 found.

18 A. Yes.

19 Q. And the the significant dose response
20 relationship was with respect to dollars spent on fuel
21 and oil for farm purposes for 1970.

22 Would you agree with me, Dr. Rodricks,
23 that during the period of exposure in this study the
24 authors indicate that 2,4-D constituted over 90 per
25 cent and 75 per cent by weight of all herbicide active

1 ingredients used agriculturally in Saskatchewan?

2 A. I would need to look that up, sir.

3 Q. Page 580, left-hand column. I guess
4 it is the second full paragraph on the page, left-hand
5 column.

6 A. I'm sorry, on page...

7 Q. 580.

8 A. Oh, sorry. I am afraid my copy is
9 missing 580. No wonder I am confused.

10 Q. That would make it difficult to find
11 it.

12 A. As a matter of it is missing every
13 other page. This was the copy I was given during
14 lunch.

15 MR. CASSIDY: This one has got them call.

16 MR. CASTRILLI: By your counsel, I would
17 note.

18 MR. CASSIDY: --my photocopying was not
19 that bad.

20 MR. CASTRILLI: Did I hear the sound of
21 an exhibit being torn up?

22 MR. CASSIDY: The witness' copy. The
23 official exhibit I believe is with Mr. Martel's
24 safekeeping.

25 DR. RODRICKS: I found the paragraph,

1 yes.

2 MR. CASTRILLI: Q. You don't have any
3 better information with respect to that study; do you?

4 DR. RODRICKS: A. Certainly not.

5 Q. And the herbicide 2,4,5-T -- sorry I
6 am just reading from the next sentence in that
7 paragraph.

8 "The herbicide 2,4,5-T was apparently
9 used infrequently in agriculture in
10 Saskatchewan at this time, although it
11 was in regular use for brush control on
12 non-crop land."

13 There are several references. Now, you
14 don't have any better information than that, I presume;
15 is that right?

16 A. No, I do not.

17 Q. Now, Dr. Rodricks, do you have
18 Exhibit 717 which was the abstract that was produced in
19 August of 1989 by Dr. Ritter with respect to this study
20 which, of course, at that time hadn't been written?

21 A. I'm afraid I don't have that
22 abstract. It is not identical to the one in the
23 report?

24 Q. That's what we are going to explore.

25 A. I do not have the abstract.

1 MADAM CHAIR: Mr. Castrilli, why are we
2 going to explore whether this abstract is the same as
3 the abstract in the published article?

4 MR. CASTRILLI: Well, for one reason,
5 because I don't believe they are the same and for a
6 second reason, Exhibit 717 has been on the record for
7 roughly 12 -- 10 months. I believe there was
8 commentary on the record with respect to the abstract
9 last year and I just want to get the record clarified.

10 Madam Chair, actually noticing that it is
11 almost five o' clock, what I am suggest to suggest is
12 that the witnesses be given a copy of the abstract
13 from -- sorry, that would be Exhibit 717.

14 Q. One other item. Dr. Rodricks, you
15 will notice if you look at the front page of Exhibit
16 1224--

17 DR. RODRICKS: A. Yes.

18 Q. --under the heading of Editorials,
19 You will see in the second listed item there is an
20 editorial by Aaron Blair, Herbicides and Non-Hodgkin's
21 Lymphoma, New Evidence From a study of Saskatchewan
22 Farmers. Do you have a copy of that?

23 A. I don't.

24 MR. CASTRILLI: Madam Chair, I would like
25 to make a copy of the item I have just referred to from

1 exhibit 1244 available to the witness for him to
2 consider overnight and I would suggest that that's
3 where we resume tomorrow morning.

4 MADAM CHAIR: All right. One thing, Mr.
5 Castrilli. Why don't you tell Dr. Rodricks what the
6 difference is that you see. It shouldn't take long to
7 clear this up.

8 MR. CASTRILLI: No, it won't. Dr.
9 Rodricks, perhaps overnight you could simply review the
10 first paragraph of Exhibit 717, which is the abstract,
11 and just advise the Board whether you find that
12 paragraph is still a conclusion of the authors or
13 whether it is no longer a conclusion of the authors as
14 reported in Exhibit 1244.

15 DR. RODRICKS: Whether the first
16 paragraph of the abstract...

17 MR. CASTRILLI: In Exhibit 717 is still a
18 part of --

19 DR. RODRICKS: The conclusions of this
20 paper.

21 MR. CASTRILLI: The conclusions from
22 Exhibit 1244. It is an exercise that should not take
23 very long. I will now make a copy available of the
24 editorial from Aaron Blair and I would suggest that
25 that is where we leave matters for this afternoon.

1 MR. CASSIDY: Do you have a copy you are
2 going to give to him right now?

3 MR. CASTRILLI: Yes, I am going to make a
4 copy available to him.

5 MR. CASSIDY: All right.

6 MADAM CHAIR: Are we going to make that
7 an exhibit, Mr. Castrilli?

8 MR. CASTRILLI: Yes.

9 MADAM CHAIR: Exhibit 1248.

10 ---EXHIBIT NO. 1248: Editorial entitled Herbicides and
11 Non-Hodgkin's Lymphoma, New
12 Evidence From a study of
Saskatchewan Farmers by Aaron
Blair.

13 MR. CASSIDY: It may be necessary for me
14 to speak to the witnesses, Madam chair, to assist them
15 in finding a copy of Exhibit 177, and that's all I
16 intend to do.

17 MADAM CHAIR: Here is my copy. You can
18 have a copy run off...

19 MR. CASSIDY: I also can advise, Madam
20 Chair, on this matter that arose over the course of
21 lunch, that prior to the completion of the
22 examination-in-chief the witnesses were asked to be
23 provided with a transcript that refers to another
24 exhibit that Mr. Castrilli -- relates to another
25 exhibit that Mr. Castrilli advises he intends to

1 cross-examine on, Exhibit 789.

2 The transcript volume is 125 and I've now
3 located it and I intend to provide that to the
4 witnesses as well.

5 MADAM CHAIR: Any objections?

6 MR. CASTRILLI: No, not at all.

7 DR. RACHMAN: Before we break, could I
8 please ask for a clarification of which materials
9 exactly you want us to look at this evening to make
10 sure that we have a complete list.

11 MR. CASTRILLI: Yes. Madam Chair, I've
12 made a number of articles available to the witnesses
13 that are not yet exhibits in which we will be
14 discussing tomorrow morning. I intend to examine them
15 on those documents.

16 With respect to Exhibit 1233, which I
17 identified earlier today as an exhibit I might wish to
18 raise with the witnesses, I can advise at this time
19 that I don't think it will necessary for the panel to
20 actually look at Exhibit 1233.

21 DR. RACHMAN: What was Exhibit 1233?

22 MR. CASTRILLI: Sorry, 1233 is the
23 phonebook in front of you.

24 DR. RACHMAN: Oh, the one I have already
25 read?

1 MR. CASTRILLI: If you've read it between
2 midday and now...

3 MADAM CHAIR: So how many different
4 pieces of documentation do they have to read tonight,
5 Mr. Castrilli?

6 MR. CASTRILLI: Well, the remaining
7 documentation has not yet been filed with the Board.
8 I've given them roughly a half a dozen pieces of paper.

9 MADAM CHAIR: Now, can you direct them
10 more carefully in their reading so they can answer
11 succinctly--

12 MR. CASTRILLI: I think the --

13 MADAM CHAIR: --tomorrow what the
14 questions are going to be.

15 MR. CASTRILLI: Well, I think they should
16 read the entirety of the documents. They are not very
17 long in most cases. Some of them are one page long.

18 DR. RACHMAN: What about 1237?

19 MR. CASTRILLI: I think we've dealt with
20 1238 already on the record today and I don't believe
21 there is anything further that needs to be referred to
22 with respect to that exhibit.

23 And just to be fair to the witnesses,
24 with respect to one document I've provided to them,
25 which is called the Record of Decision, Pacific

1 Northwest, they need only look at page 6.

2 MR. CASSIDY: That's Exhibit 1236, I
3 think.

4 MR. CASSIDY: No, that's another one I
5 have just given them--

6 MR. CASSIDY: Excuse me.

7 MR. CASTRILLI: --that is not yet an
8 exhibit.

9 DR. RACHMAN: Page 6 only?

10 MR. CASTRILLI: Page 6 of the Record of
11 Decision, Pacific Northwest.

12 Everything else is comparatively short,
13 Madam Chair. There is one item that's a little bit
14 longer, but it is pretty straightforward and they won't
15 have much difficulty with it.

16 I would like at this time to make the
17 Aaron Blain editorial the next exhibit.

18 MADAM CHAIR: Thank you. We said that
19 would be Exhibit 1248.

20 MR. CASTRILLI: Madam Chair, we will be
21 resuming at 8:30 tomorrow morning?

22 MADAM CHAIR: Yes. When will you be
23 finished your cross-exam, Mr. Castrilli?

24 MR. CASTRILLI: Madam Chair, I anticipate
25 being completed by the midday break, lunch time.

1 MADAM CHAIR: Lunch time.

2 MR. CASTRILLI: Thereabouts.

3 MADAM CHAIR: All right. And Ms. Kleer
4 is to follow Mr. Castrilli, so she should be here by...

5 MR. CASTRILLI: I would say late morning
6 at the earliest. Thank you, witnesses.

7 MADAM CHAIR: Mr. Freidin?

8 MR. FREIDIN: Madam Chair, I am just
9 wondering if I could just address the Board for a
10 moment, just take a couple of moments.

11 I assume that the Board has heard about
12 the announcements by the government last week that Dr.
13 Peter Pearce of the University of British Columbia has
14 been retained by the government to perform certain work
15 which, I understand, will lead to certain
16 recommendations to the Minister of Natural Resources
17 concerning an appropriate process for the development
18 of an overall forest policy for Ontario.

19 MADAM CHAIR: I haven't heard anything
20 about this, Mr. Freidin.

21 MR. FREIDIN: All right. On that basis,
22 that's why I rose, I felt that as a courtesy to the
23 Board I should bring this to the Board's attention and
24 I would like to provide you with certain documentation
25 which was released along with that announcement, not to

1 suggest that you don't have enough reading to do as it
2 is.

3 I don't believe these need be marked
4 exhibits, Madam Chair, but the documents I would like
5 to give you and Mr. Martel are as follows: The
6 curriculum vitae of Dr. Pearce, the terms of reference
7 for Dr. Pearce dated May the 10th, 1990, and a letter
8 from the Minister. It is standard form letter from the
9 Minister of Natural Resources which was sent out to a
10 large number of groups and individuals, and I can
11 assure you that that large group included parties at
12 this particular proceeding.

13 MADAM CHAIR: So give it to us in a
14 nutshell, Mr. Freidin, what does all of this mean?

15 MR. CASTRILLI: Madam Chair, I wonder if
16 Mr. Freidin could advise when this was released to the
17 public?

18 MR. FREIDIN: I believe on the day it
19 indicated on the terms of reference. I think May the
20 10th. I can't be precise about -- I would have to
21 check on the precise date.

22 The reason I provided this to the Board,
23 Madam Chair, is that it is a matter of -- it may be a
24 matter of interest to the Board as it is dealing with a
25 related or complementary matter; that is, an overall

1 policy for forestry in Ontario, it is not limited to
2 timber management at all.

3 The reason I wanted to bring it to your
4 attention is that I felt if you heard about it sort of
5 via grapevine you might ask the question as to what
6 relationship, if any, it has to what the Board is
7 doing, whether it affects your ability to do all the
8 things that we have indicated in past submissions the
9 Board can do. I didn't want that question to sort of
10 come to your mind if you heard about it through the
11 grapevine and wonder what the answer to that was.

12 So I just wanted to provide you with the
13 material that I have and perhaps just refer you to the
14 letter from the Minister which indicates that it is
15 certainly the Ministry's view, on page 2, if you look
16 at the fourth paragraph and the first sentence, that's
17 all I need refer to, the Minister has stated that she
18 wants to emphasize to the people that -- she is
19 advising about this development. She wants to
20 emphasize that this policy and review will not
21 duplicate or pre-empt the ongoing environmental
22 assessment dealing with timber management.

23 So, again, I just felt as a matter of
24 courtesy I should bring this to your attention and
25 answer a question which I thought might come to your

1 mind if you had heard about it, and I believe perhaps a
2 review of the material that I provided may be of
3 assistance.

4 MADAM CHAIR: Thank you, Mr. Freidin.

5 MR. HUFF: Mr. Freidin, could I have a
6 copy of the press release that went out last Wednesday
7 afternoon?

8 MR. FREIDIN: I don't have a copy of the
9 press release.

10 MR. HUFF: Can I have it tomorrow if you
11 can get it?

12 MR. FREIDIN: Sure.

13 MADAM CHAIR: All right. We will adjourn
14 until tomorrow morning at 8:30. Thank you.

15

16 ---Whereupon the hearing adjourned at 5:10 p.m.,
17 to be reconvened Thursday, June 14, 1990 commencing
at 8:30 a.m.

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